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NEWS 2	Aug 21	CAS patent coverage expanded
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NEWS 4	Aug 28	MEDLINE from 1958 to Date - Only on STN
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NEWS 7	Sep 21	KKF renamed DKILIT
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NEWS 11	Oct 27	Patent Assignee Code Dictionary now available in Derwent Patent Files
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FILE 'HOME' ENTERED AT 12:41:35 ON 03 NOV 2000

=> file hcaold

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all substance data from the REGISTRY file.  Enter HELP FIRST for
more information.

=> e reed-gitomer,b/au

E1          10     REED WINSTON H/AU
E2           1     REED X B JR/AU
E3           0 --> REED-GITOMER,B/AU
E4           3     REEDAL J S/AU
E5           1     REEDER A H/AU
E6           1     REEDER A M/AU
E7           1     REEDER A S/AU
E8           1     REEDER C/AU
E9           2     REEDER C E/AU
E10          2     REEDER D B/AU
E11          2     REEDER D D/AU
E12         19     REEDER E/AU

=> e pak, c/au

E1           6     PAK YA V/AU
E2           3     PAK Z P/AU
E3           0 --> PAK, C/AU
E4           1     PAKA J/AU
E5           3     PAKALNE D/AU
E6           1     PAKALNET YA F/AU
E7           3     PAKALNS G/AU
E8           5     PAKALNS P/AU
E9           7     PAKANAEV YA I/AU
E10          7     PAKARSKYTE K/AU
E11          1     PAKAULLA I/AU
E12          2     PAKCHANIN L M/AU

=> file registry

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                                                 ENTRY     SESSION
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TSCA INFORMATION NOW CURRENT THROUGH July 8, 2000

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=> e hypercalciuria

E1	186		HYPERCALCEMIC/BI
E2	2		HYPERCALCI/BI
E3	2	-->	HYPERCALCIURIA/BI
E4	3		HYPERCALIN/BI
E5	2		HYPERCARB/BI
E6	2		HYPERCHLOR/BI
E7	2		HYPERCHLORITO/BI
E8	1		HYPERCO/BI
E9	1		HYPERCONFLUENTIC/BI
E10	7		HYPERCRATINE/BI
E11	1		HYPERCYL/BI
E12	8		HYPERD/BI

=> s e3

L1 2 HYPERCALCIURIA/BI

=> file hcaold

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	4.20	16.95

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FILE COVERS 1907-1966
FILE LAST UPDATED: 01 May 1997 (19970501/UP)

This file contains CAS Registry Numbers for easy and accurate substance identification. Title keywords, authors, patent assignees, and patent information, e.g., patent numbers, are now searchable from 1907-1966. TIFF images of CA abstracts printed between 1907-1966 are available in the PAGE display formats.

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=> s hypercalciuria

L2 19 HYPERCALCIURIA

=> s l1 and l2

0 L1

```
L3            0 L1 AND L2

=> s l2 and screening

          1225 SCREENING
L4           0 L2 AND SCREENING

=> s l2 and (detection method or screening or testing or risk assessment or
risk?)

         12847 DETECTION
         22157 METHOD
            15 DETECTION METHOD
                  (DETECTION(W)METHOD)
          1225 SCREENING
         10106 TESTING
            69 RISK
           505 ASSESSMENT
             0 RISK ASSESSMENT
                  (RISK(W)ASSESSMENT)
           140 RISK?
L5           0 L2 AND (DETECTION METHOD OR SCREENING OR TESTING OR RISK
ASSESSM
               ENT OR RISK?)

=> s l2 and (risk or increased risk or risk assessment?)

            69 RISK
          2254 INCREASED
            69 RISK
             0 INCREASED RISK
                  (INCREASED(W)RISK)
            69 RISK
           515 ASSESSMENT?
             0 RISK ASSESSMENT?
                  (RISK(W)ASSESSMENT?)
L6           0 L2 AND (RISK OR INCREASED RISK OR RISK ASSESSMENT?)

=> d l2 ti abs ibib tot

'ABS' IS NOT A VALID FORMAT FOR FILE 'HCAOLD'

The following are valid formats:

ALL ----- AN, TI, AU, PA, DT, IT, PI (default)
BIB ----- AN, TI, AU, PA, DT, PI
CAN ----- List of CA abstract numbers, no L-number headers
CBIB ---- AN, TI, AU, PA, PI
DALL ---- ALL, delimited (end of each field identified)
IND ----- Indexing data
MAX ----- Same as ALL
SAM ----- TI, IT
SCAN ---- TI, IT (random display, no answer numbers;
          SCAN must be entered on the same line as the DISPLAY,
          e.g., D SCAN or DISPLAY SCAN)
STD ----- BIB

IALL ---- ALL, indented with text labels
IBIB ---- BIB, indented with text labels
ISTD ---- STD, indented with text labels

HIT ----- Fields containing hit terms
HITIND -- IT
```

HITRN --- HIT RN
HITSTR -- HIT RN, its CA index name and its structure diagram
FHITSTR - First HIT RN, its CA index name and its structure diagram
OCC ----- Number of occurrence of hit term and fie ld in which it occurs

Index Terms (IT) are CAS Registry Numbers; Accession
Numbers (AN) CA References.

Index Terms in CAOLD include only Registry Numbers; no
subject terms are available. The same formats (except
SAMPLE) may be used with the DISPLAY ACC command to display
the record for a specified CAOLD Accession Number.

PAGE ---- Page Image of original Chemical Abstracts issue containing the
abstract of the answer.
PAGE.PREV and PAGE.NEXT will return the image of the page before or after
the current answer.
ENTER DISPLAY FORMAT (ALL):end

=> d l2 all

L2 ANSWER 1 OF 19 HCAOLD COPYRIGHT 2000 ACS
AN CA64:16506h CAOLD
TI effects of thiazides in idiopathic **hypercalciuria**
AU Yendt, Edmund R.; Gagne, R. J. A.; Cohanim, M.

=> d l2 ti abs tot

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The following are valid formats:

ALL ----- AN, TI, AU, PA, DT, IT, PI (default)
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SAM ----- TI, IT
SCAN ---- TI, IT (random display, no answer numbers;
SCAN must be entered on the same line as the DISPLAY,
e.g., D SCAN or DISPLAY SCAN)
STD ----- BIB

IALL ---- ALL, indented with text labels
IBIB ---- BIB, indented with text labels
ISTD ---- STD, indented with text labels

HIT ----- Fields containing hit terms
HITIND -- IT
HITRN --- HIT RN
HITSTR -- HIT RN, its CA index name and its structure diagram
FHITSTR - First HIT RN, its CA index name and its structure diagram
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Numbers (AN) CA References.

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PAGE ---- Page Image of original Chemical Abstracts issue containing the abstract of the answer.
PAGE.PREV and PAGE.NEXT will return the image of the page before or after the current answer.
ENTER DISPLAY FORMAT (ALL):end

=> d l2 ti tot

L2 ANSWER 1 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI effects of thiazides in idiopathic **hypercalciuria**

L2 ANSWER 2 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI metabolic studies in patients with idiopathic **hypercalciuria**

L2 ANSWER 3 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI altering the effect of adrenaline with caffeine
TI effect of adrenocortical steroids on the **hypercalciuria** of Wilson's disease

L2 ANSWER 4 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI idiopathic **hypercalciuria** in a child

L2 ANSWER 5 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI comparison of the liver-protecting ability of some derivs. of betaine, choline, and methionine
TI toxic action of vitamin D in the absence of hypercalcemia in parathyroidectomized rats - (II) action of the Ca level in **hypercalciuria**
TI toxic action of vitamin D in the absence of hypercalcemia, in parathyroidectomized rats - (I) anat. lesions

L2 ANSWER 6 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI relation between citric acid and Ca metabolism-primary hyperparathyroidism and idiopathic **hypercalciuria**

L2 ANSWER 7 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI idiopathic **hypercalciuria**, pathogenic and therapeutic study

L2 ANSWER 8 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI kidney stones resulting from **hypercalciuria**

L2 ANSWER 9 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI impairment of renal concg. ability in prolonged hypercalcemia and **hypercalciuria**

L2 ANSWER 10 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI factors intervening in atherosclerosis
TI idiopathic **hypercalciuria**

L2 ANSWER 11 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI **hypercalciuria** in sarcoidosis, idiopathic **hypercalciuria**, and that produced by vitamin D-Ca metabolism

L2 ANSWER 12 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI induced **hypercalciuria** as an indicator of bone metabolism

L2 ANSWER 13 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI amelioration of **hypercalciuria** following poliomyelitis by 17-ethyl-19-nort[redacted]osterone

L2 ANSWER 14 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI effect of therapeutic mobilization on **hypercalciuria** following acute poliomyelitis

L2 ANSWER 15 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI CO2 anesthesia and reversible suppression of the effects of urethan anesthesia by CO2 in dogfish
TI hypocalcemic **hypercalciuria** during vitamin D and dihydrotachysterol therapy of hypoparathyroidism

L2 ANSWER 16 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI **hypercalciuria** following poliomyelitis-relation to site and degree of paralysis

L2 ANSWER 17 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI Idiopathic **hypercalciuria**

L2 ANSWER 18 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI Syndromes of alimentary osteomalacia and osteoporosis - (I) static syndromes, (II) **hypercalciuria**, (III) vitamin D modification of the calciuria, (IV) limits of diagnostic value of dynamic skeleton explosion

L2 ANSWER 19 OF 19 HCAOLD COPYRIGHT 2000 ACS
TI Etiology and treatment of kidney stones
TI Idiopathic **hypercalciuria**

=> file biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	19.66	36.61

FILE 'BIOSIS' ENTERED AT 12:51:33 ON 03 NOV 2000
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 1 November 2000 (20001101/ED)

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=> s l1

L7 0 L1

=> d his

(FILE 'HOME' ENTERED AT 12:41:35 ON 03 NOV 2000)

FILE 'HCAOLD' ENTERED AT 12:41:54 ON 03 NOV 2000
E REED-GITOMER,B/AU
E PAK, C/AU

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     FILE 'REGISTRY' ENTERED AT 12:45:48 ON 03 NOV 2000
                E HYPERCALCIURIA
L1             2 S E3

     FILE 'HCAOLD' ENTERED AT 12:46:53 ON 03 NOV 2000
L2            19 S HYPERCALCIURIA
L3             0 S L1 AND L2
L4             0 S L2 AND SCREENING
L5             0 S L2 AND (DETECTION METHOD OR SCREENING OR TESTING OR RISK
ASSE
L6             0 S L2 AND (RISK OR INCREASED RISK OR RISK ASSESSMENT?)

     FILE 'BIOSIS' ENTERED AT 12:51:33 ON 03 NOV 2000
L7             0 S L1

=> s hypercalciuria

L8          1896 HYPERCALCIURIA

=> s l8 and (method of screening or risk or risk assessment or detection?)

         1482337 METHOD
          103271 SCREENING
            2608 METHOD OF SCREENING
                   (METHOD(1W)SCREENING)
          259383 RISK
          259383 RISK
          317478 ASSESSMENT
           10148 RISK ASSESSMENT
                   (RISK(W)ASSESSMENT)
          266790 DETECTION?
L9           177 L8 AND (METHOD OF SCREENING OR RISK OR RISK ASSESSMENT OR
DETECT
                 ION?)

=> s l9 and (increased risk?)

          961477 INCREASED
          273131 RISK?
           20440 INCREASED RISK?
                   (INCREASED(W)RISK?)
L10            5 L9 AND (INCREASED RISK?)

=> d l10 ti abs ibib tot


L10  ANSWER 1 OF 5  BIOSIS  COPYRIGHT 2000 BIOSIS
TI   Course and outcome of pregnancy in a patient with mild, asymptomatic,
     primary hyperparathyroidism diagnosed before conception.
AB   Primary hyperparathyroidism (PHP) during pregnancy is well known to
confer
     an increased risk of complications to both the mother
     and the fetus. However, the risks and optimal management of patients with
     mild, asymptomatic disease during pregnancy are much less clear. We
     observed a patient with mild, asymptomatic PHP who was diagnosed before
     conception through pregnancy. The patient remained asymptomatic through
     the first 22 weeks of pregnancy, and her calcium levels remained under 11
     mg/dL. This occurred despite a dramatic elevation in the level of
     1,25-dihydroxyvitamin D and marked hypercalciuria. Parathyroid
     surgery was performed at 22 weeks of gestation and a parathyroid adenoma
     was removed. Postoperatively, the patient's calcium level normalized and
     the rest of the pregnancy was uncomplicated. The patient delivered a
     healthy baby at 40 weeks of gestation. The neonatal course was
```

unremarkable. We conclude that mild, asymptomatic PHP during early pregnancy is compatible with normal fetal development and an uncomplicated pregnancy and that the serum calcium level in such patients can remain stable with medical management alone, despite the marked changes in maternal calcium metabolism that characterize normal pregnancy.

ACCESSION NUMBER: 2000:432351 BIOSIS
DOCUMENT NUMBER: PREV200000432351
TITLE: Course and outcome of pregnancy in a patient with mild, asymptomatic, primary hyperparathyroidism diagnosed before conception.
AUTHOR(S): Tollin, Steven R. (1)
CORPORATE SOURCE: (1) 4000 Presidential Blvd., Lincoln Green Apartments, Apt. 1510, Philadelphia, PA, 19131 USA
SOURCE: American Journal of the Medical Sciences, (August, 2000) Vol. 320, No. 2, pp. 144-147. print. ISSN: 0002-9629.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

L10 ANSWER 2 OF 5 BIOSIS COPYRIGHT 2000 BIOSIS
TI Effect of calcitriol on bone loss after cardiac or lung transplantation.
AB Rapid bone loss after cardiac and lung transplantation results in an **increased risk** of osteoporotic fracture. This study examined the efficacy of treatment with calcitriol (1,25-dihydroxyvitamin D3) in preventing bone loss in patients undergoing cardiac or lung transplantation. In this 2-year double-blind, stratified study, 65 patients undergoing cardiac or single lung transplantation were randomly allocated to receive either placebo or calcitriol (0.5-0.75 mg/day), the latter for either 12 months or 24 months. All patients received 600 mg calcium/day. Bone mineral density (BMD) was measured every 6 months for 2 years by dual-energy X-ray absorptiometry. There was no significant difference between groups with respect to age or cumulative dose of prednis(ol)one or cyclosporine over the 2 years. Bone loss at the proximal femur was significantly reduced or prevented at all three sites by treatment with calcitriol for 2 years compared with treatment with calcium alone. Treatment with calcitriol for 12 months followed by calcium for 12 months resulted in similar proximal femoral bone loss to that seen in those patients treated with calcium for 24 months, suggesting calcitriol prophylaxis needs to be continued beyond 12 months. At the lumbar spine, there were no significant differences in BMD between groups. Over a period of 2 years, 22 new vertebral fractures/deformities occurred in 4 patients treated with calcium alone compared with one new vertebral fracture in 1 patient treated with calcitriol. Because the sample size was too low to provide reliable interpretation of vertebral fracture rates, this difference is likely a chance result. Mild hypercalcemia was common with calcitriol therapy, as was mild **hypercalciuria** (59% of patients vs. 10% controls), but there were no significant differences between groups in serum creatinine after 2 years. These data suggest calcitriol has a role in reducing proximal femur bone loss after cardiac or lung transplantation but treatment needs to be continued beyond 1 year.

ACCESSION NUMBER: 2000:424927 BIOSIS
DOCUMENT NUMBER: PREV200000424927
TITLE: Effect of calcitriol on bone loss after cardiac or lung transplantation.
AUTHOR(S): Sambrook, Philip (1); Henderson, N. Kathy; Keogh, Anne; Macdonald, Peter; Glanville, Allan; Spratt, Phillip; Bergin, Peter; Ebeling, Peter; Eisman, John

CORPORATE SOURCE: (1) Institute of Bone and Joint Research, University of Sydney, Sydney, 2065 Australia
SOURCE: Journal of Bone and Mineral Research (September, 2000) Vol. 15, No. 9, pp. 1818-1824. print.
ISSN: 0884-0431.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

L10 ANSWER 3 OF 5 BIOSIS COPYRIGHT 2000 BIOSIS
TI Unravelling the links between calcium excretion, salt intake, hypertension, kidney stones and bone metabolism.
AB Evidence from animal, clinical and epidemiological studies suggests that high blood pressure is associated with abnormalities of calcium metabolism, leading to increased calcium loss, secondary activation of the parathyroid gland, increased movement of calcium from bone and **increased risk** of urinary tract stones. Some of these abnormalities are detectable in children and young people and continue throughout adult life. The cluster of abnormalities may be due either to a primary renal tubular defect ('renal calcium leak' hypothesis) or to the effect of central volume expansion seen in hypertension ('central blood volume' hypothesis). A high salt intake is known to aggravate these abnormalities and their consequences. If substantial calcium loss related to high blood pressure is sustained over many decades, increased excretion of calcium in the urine may result in an **increased risk** of urinary tract stones, and the increased movement of calcium from bone may result in higher rates of bone mineral loss, thereby increasing the **risk** of osteoporosis. The present review summarises the evidence, suggests a unifying hypothesis and discusses clinical and public health implications.
ACCESSION NUMBER: 2000:350827 BIOSIS
DOCUMENT NUMBER: PREV200000350827
TITLE: Unravelling the links between calcium excretion, salt intake, hypertension, kidney stones and bone metabolism.
AUTHOR(S): Cappuccio, Francesco P. (1); Kalaitzidis, Rigas; Duneclift, Stuart; Eastwood, John B.
CORPORATE SOURCE: (1) Department of Medicine, St George's Hospital Medical School, Cranmer Terrace, London, SW17 0RE UK
SOURCE: JN Journal of Nephrology, (May June, 2000) Vol. 13, No. 3, pp. 169-177. print.
ISSN: 1121-8428.
DOCUMENT TYPE: General Review
LANGUAGE: English
SUMMARY LANGUAGE: English

L10 ANSWER 4 OF 5 BIOSIS COPYRIGHT 2000 BIOSIS
TI EFFECTS OF DIFFERENT DOSES OF ALKALINE CITRATE ON URINE COMPOSITION AND CRYSTALLIZATION OF CALCIUM OXALATE.
AB Prophylactic treatment with alkaline citrate in patients with recurrent calcium oxalate (CaOx) stone disease results in reduced CaOx supersaturation and increased urinary citrate. The effects of a single evening dose were compared with those of two and three daily doses in six recurrent CaOx stone formers with **hypercalciuria**, hypocitraturia or raised calcium/citrate quotients. While on a standardized hospital diet the patients were given 7.5 g (28 mmol) of sodium potassium citrate (URALYT-U) in one, two, and three doses. Fractional urine collections during 24 hours were analyzed for pH, composition, and crystallization **risk** (CR). All dosage regimens had favourable effects on urinary

calcium, citrate, calcium/citrate quotients, and CaOx-CR. The most sustained effect doses resulted in the most pronounced effects between 22.00-06.00 h, t[redacted]eby counteracting the **increase[redacted]isk** of CaOx crystallization during that period. In terms of 24 h urine composition the best effect was recorded with alkaline citrate administered three times daily, but because of the favourable response by a single evening dose between 22.00-06.00 h the assumption was made that this dosage regimen might be sufficient to reduce the **risk** of CaOx crystallization and stone formation. However, the validity of such an assumption can only be established by long-term clinical studies.

ACCESSION NUMBER: 1990:201877 BIOSIS
DOCUMENT NUMBER: BA89:108548
TITLE: EFFECTS OF DIFFERENT DOSES OF ALKALINE CITRATE ON URINE COMPOSITION AND CRYSTALLIZATION OF CALCIUM OXALATE.
AUTHOR(S): BERG C; LARSSON L; TISELIUS H-G
CORPORATE SOURCE: DEP. UROL., UNIV. HOSP., S-58185 LINKOPING, SWEDEN.
SOURCE: UROL RES, (1990) 18 (1), 13-16.
CODEN: URLRA5. ISSN: 0300-5623.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L10 ANSWER 5 OF 5 BIOSIS COPYRIGHT 2000 BIOSIS
TI CALCIUM OXALATE KIDNEY STONES IN PATIENTS ON CONTINUOUS AMBULATORY PERITONEAL DIALYSIS.
AB Kidney stones were passed by 10 out of 186 patients with endstage renal disease who were treated with continuous ambulatory peritoneal dialysis (CAPD). Stones from 7 patients were examined by x-ray diffraction. In 5 of them, the stones were composed of calcium oxalate monohydrate. The urine calcium oxalate activity product was determined in 44 CAPD patients, 8 of whom were stone formers, and compared to that of 120 normal volunteers. In CAPD patients, mean urine Ca+ concentration was lower than in normal subjects whereas mean urine ionic oxalate concentration was significantly higher than in normal subjects. In normal urine samples, the calcium oxalate activity product showed a significant correlation with both the urine Ca+ and the ionic oxalate concentrations. In CAPD patients, the calcium oxalate activity product correlated with the Ca+ concentration but not with ionic oxalate. Although the urine Ca+ concentrations is lower in CAPD patients than in normal subjects, it is the relative increase in its concentration which appears to be associated with the **increased risk** of kidney stone formation in these patients. This relative **hypercalciuria** seems to follow 1,25(OH)2 vitamin D3 administration.

ACCESSION NUMBER: 1984:303618 BIOSIS
DOCUMENT NUMBER: BA78:40098
TITLE: CALCIUM OXALATE KIDNEY STONES IN PATIENTS ON CONTINUOUS AMBULATORY PERITONEAL DIALYSIS.
AUTHOR(S): OREN A; HUSDAN H; CHENG P-T; KHANNA R; PIERRATOS A; DIGENIS G; OREOPOULOS D G
CORPORATE SOURCE: DIV. NEPHROL., SUITE 525, TORONTO WESTERN HOSP., 399 BATHURST ST., TORONTO, ONTARIO M5T 2S8, CANADA.
SOURCE: KIDNEY INT, (1984) 25 (3), 534-538.
CODEN: KDYIA5. ISSN: 0085-2538.
FILE SEGMENT: BA; OLD
LANGUAGE: English

=> s l8 and genetic mutation

```
        337033 GENETIC
        117015 MUTATION
          1299 GENETIC MUTATION
                 (GENETIC(W)MUTATION)
L11           1 L8 AND GENETIC MUTATION

=> d l11 ti abs ibib tot


L11  ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2000 BIOSIS
TI   Familial benign hypercalcemia with hypercalciuria in the elderly
     members of the family due to 3q mutation.
ACCESSION NUMBER:    1999:324345  BIOSIS
DOCUMENT NUMBER:     PREV199900324345
TITLE:               Familial benign hypercalcemia with hypercalciuria
                     in the elderly members of the family due to 3q mutation.
AUTHOR(S):           Soei, Y. L. (1); Karperien, M. (1); Bakker, B.; Breuning,
                     M. H.; Hendy, G. N.; Papapoulos, S. E. (1)
CORPORATE SOURCE:    (1) Department of Endocrinology, Leiden University Medical
                     Center, Leiden Netherlands
SOURCE:              Calcified Tissue International, (1999) Vol. 64, No. SUPPL.
                     1, pp. S86.
                     Meeting Info.: XXVIth European Symposium on Calcified
                     Tissues Maastricht, Netherlands May 7-11, 1999 European
                     Calcified Tissue Society
                     . ISSN: 0171-967X.
DOCUMENT TYPE:       Conference
LANGUAGE:            English


=> file hcaplus

COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                               15.39      52.00

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26, 1996), unless otherwise indicated in the original publications.

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FILE LAST UPDATED: 2 Nov 2000` (20001102/ED)

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this searchable data in CAOLD.  You now have electronic access to all
of CA: 1907 to 1966 in CAOLD and 1967 to the present in HCAPLUS on STN.

=> s l8
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L12         662 HYPERCALCIURIA

=> s l12 and mutation

        141810 MUTATION
L13         44 L12 AND MUTATION

=> s l13 and chromosome 1

         93167 CHROMOSOME
       5511960 1
          3124 CHROMOSOME 1
                 (CHROMOSOME(W)1)
L14          1 L13 AND CHROMOSOME 1

=> d l14 ti abs ibib tot


L14  ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2000 ACS
TI   Absorptive hypercalciuria locus on human chromosome
     1
AB   Disclosed is a region on human chromosome 1 that
     provides a genetic basis for absorptive hypercalciuria and thus
     to some forms of osteoporosis as well.  The region is defined as contg.
     1q23 and 1q24, a 4.3 megabase region between markers D1S2681 and D1S2815.
     A 2567-bp cDNA encoding a hypothetical protein of unknown function was
     mapped to this genomic region, and the genomic sequence reveals a gene of
     at least 38,844 bp encompassing at least 16 exons.  The genes, proteins,
     and other biol. materials provided are envisioned for use in diagnostic
     and therapeutic methods related to absorptive hypercalciuria and
     osteoporosis with hypercalciuria.
ACCESSION NUMBER:         1999:819532  HCAPLUS
DOCUMENT NUMBER:          132:74536
TITLE:                    Absorptive hypercalciuria locus on human
                          chromosome 1
INVENTOR(S):              Reed-Gitomer, Berenice Y.; Pak, Charles Y. C.
PATENT ASSIGNEE(S):       Board Or Regents, the University of Texas System, USA
SOURCE:                   PCT Int. Appl., 153 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 9967426         A1   19991229       WO 1999-US14347  19990623
         W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ,
             DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS,
             JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,
             MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,
             TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ,
             MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
             ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,
             CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     AU 9948310         A1   20000110       AU 1999-48310    19990623
PRIORITY APPLN. INFO.:                     US 1998-90348    19980623
                                           WO 1999-US14347  19990623

REFERENCE COUNT:          5
REFERENCE(S):             (1) Imamura; American Journal of Medical Genetics
                              1998, V17(1), P52
```

Appl

(2) Lemann; Journal of Urology 1989, V141, P715
(3) Mullis; US 4683202 A 1987
(4) Pearce; The New England Journal of Medicine 1996, V335(15), P1122
(5) Thakker, R; Nova Acta Leopolidina 1997, V75(302), P23 HCAPLUS

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=> d his

     (FILE 'HOME' ENTERED AT 12:41:35 ON 03 NOV 2000)

     FILE 'HCAOLD' ENTERED AT 12:41:54 ON 03 NOV 2000
               E REED-GITOMER,B/AU
               E PAK, C/AU

     FILE 'REGISTRY' ENTERED AT 12:45:48 ON 03 NOV 2000
               E HYPERCALCIURIA
L1             2 S E3

     FILE 'HCAOLD' ENTERED AT 12:46:53 ON 03 NOV 2000
L2            19 S HYPERCALCIURIA
L3             0 S L1 AND L2
L4             0 S L2 AND SCREENING
L5             0 S L2 AND (DETECTION METHOD OR SCREENING OR TESTING OR RISK
ASSE
L6             0 S L2 AND (RISK OR INCREASED RISK OR RISK ASSESSMENT?)

     FILE 'BIOSIS' ENTERED AT 12:51:33 ON 03 NOV 2000
L7             0 S L1
L8          1896 S HYPERCALCIURIA
L9           177 S L8 AND (METHOD OF SCREENING OR RISK OR RISK ASSESSMENT OR
DET
L10            5 S L9 AND (INCREASED RISK?)
L11            1 S L8 AND GENETIC MUTATION

     FILE 'HCAPLUS' ENTERED AT 12:55:37 ON 03 NOV 2000
L12          662 S L8
L13           44 S L12 AND MUTATION
L14            1 S L13 AND CHROMOSOME 1

=> s l13 and (screening or risk or increase risk or risk assessment?)

         91981 SCREENING
         66154 RISK
       1018679 INCREASE
         66154 RISK
            94 INCREASE RISK
                 (INCREASE(W)RISK)
         66154 RISK
         93809 ASSESSMENT?
         10773 RISK ASSESSMENT?
                 (RISK(W)ASSESSMENT?)
L15            8 L13 AND (SCREENING OR RISK OR INCREASE RISK OR RISK
ASSESSMENT?)

=> d l15 ti abs ibib tot

L15  ANSWER 1 OF 8  HCAPLUS  COPYRIGHT 2000 ACS
TI   Isolated hypercalciuria with mutation in CLCN5:
     relevance to idiopathic hypercalciuria
```

AB Idiopathic **hypercalciuria** (IH) is the most common **risk** factor for kidney stones and often has a genetic component. Dent's disease (X-linked nephrolithiasis) is assocd. with mutations in the CLCN5 chloride channel gene, and low mol. wt. (LMW) proteinuria was universally obsd. in affected males. The authors sought to identify mutations in CLCN5 or abnormalities in LMW protein excretion in a large group of patients with IH and in a rat model of genetic **hypercalciuria**. One hundred and seven patients with IH (82 adults and 25 children) and one asymptomatic hypercalciuric man with a known inactivating **mutation** in CLCN5 were studied. Secondary causes of **hypercalciuria** were excluded in all. The excretion of retinol-binding protein and .beta.2-microglobulin was measured by immunoassay in 101 patients with IH. **Mutation** anal. of the CLCN5 gene was performed in 32 patients with IH and in the genetic hypercalciuric stone-forming (GHS) rat strain. LMW protein excretion was normal in 92 patients with IH, and only slight abnormalities were found in the other nine, none of whom had a **mutation** in CLCN5. One 27-yr-old man who had a CLCN5 **mutation** was found to have isolated **hypercalciuria** without LMW proteinuria, renal failure, or other evidence of renal disease. **Mutation** anal. was normal in 32 patients with IH. The CLCN5 sequence was normal in the GHS rat. Thus, inactivation of CLCN5 can be found in the setting of **hypercalciuria** without other features of X-linked nephrolithiasis. However, mutations in CLCN5 do not represent a common cause of IH.

ACCESSION NUMBER: 2000:297697 HCAPLUS
DOCUMENT NUMBER: 133:206327
TITLE: Isolated **hypercalciuria** with **mutation** in CLCN5: relevance to idiopathic **hypercalciuria**
AUTHOR(S): Scheinman, Steven J.; Cox, Jeremy P. D.; Lloyd, Sarah E.; Pearce, Simon H. S.; Salenger, Page V.; Hoopes, Richard R.; Bushinsky, David A.; Wrong, Oliver; Asplin, John R.; Langman, Craig B.; Norden, Anthony G. W.; Thakker, Rajesh V.
CORPORATE SOURCE: Department of Medicine, SUNY Health Science Center, Syracuse, NY, USA
SOURCE: Kidney Int. (2000), 57(1), 232-239
CODEN: KDYIA5; ISSN: 0085-2538
PUBLISHER: Blackwell Science, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 47
REFERENCE(S): (1) Akuta, N; Kidney Int 1997, V52, P911 HCAPLUS
(3) Bushinsky, D; J Clin Invest 1988, V82, P1585 HCAPLUS
(4) Bushinsky, D; Kidney Int 1995, V48, P1705 HCAPLUS
(5) Bushinsky, D; Kidney Int 1999, V55, P234 HCAPLUS
(11) Devuyst, O; Hum Mol Genet 1999, V8, P247 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2000 ACS
TI Channelopathies of inwardly rectifying potassium channels
AB A review, with 104 refs. Mutations in genes encoding ion channels have increasingly been identified to cause disease conditions collectively termed channelopathies. Recognizing the mol. basis of an ion channel disease has provided new opportunities for **screening**, early diagnosis, and therapy of such conditions. This synopsis provides an overview of progress in the identification of mol. defects in inwardly

rectifying potassium (Kir) channels. Structurally and functionally distinct from other channel families, Kir channels are ubiquitously expressed and serve functions as diverse as regulation of resting membrane potential, maintenance of K+ homeostasis, control of heart rate, and hormone secretion. In humans, persistent hyperinsulinemic hypoglycemia of infancy, a disorder affecting the function of pancreatic .beta. cells, and Bartter's syndrome, characterized by hypokalemic alkalosis, **hypercalciuria**, increased serum aldosterone, and plasma renin activity, are the two major diseases linked so far to mutations in a Kir channel or assocd. protein. In addn., the weaver phenotype, a neurol. disorder in mice, has also been assocd. with mutations in a Kir channel subtype. Further genetic linkage anal. and full understanding of the consequence that a defect in a Kir channel would have on disease pathogenesis are among the priorities in this emerging field of mol. medicine.

ACCESSION NUMBER: 1999:725469 HCAPLUS
DOCUMENT NUMBER: 132:48291
TITLE: Channelopathies of inwardly rectifying potassium channels
AUTHOR(S): Abraham, M. Roselle; Jahangir, Arshad; Alekseev, Alexey E.; Terzic, Andre
CORPORATE SOURCE: Division of Cardiovascular Diseases, Department of Internal Medicine and Pharmacology, Mayo Clinic, Mayo Foundation, Rochester, MN, 55905, USA
SOURCE: FASEB J. (1999), 13(14), 1901-1910
CODEN: FAJOEC; ISSN: 0892-6638
PUBLISHER: Federation of American Societies for Experimental Biology
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 90
REFERENCE(S):
(1) Aguilar-Bryan, L; Physiol Rev 1998, V78, P227 HCAPLUS
(2) Aguilar-Bryan, L; Science 1995, V268, P423 HCAPLUS
(3) Alekseev, A; J Gen Physiol 1998, V111, P381 HCAPLUS
(4) Alekseev, A; J Membr Biol 1997, V159, P161 HCAPLUS
(5) Ashcroft, F; Trends Neurosci 1998, V21, P288 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2000 ACS
TI Functional characterization of renal chloride channel, CLCN5, mutations associated with Dent'sJapan disease
AB The annual urinary **screening** of Japanese children above three years of age has identified a progressive renal tubular disorder characterized by low mol. wt. proteinuria, **hypercalciuria** and nephrocalcinosis, and this represents a variant of Dent's disease. Hitherto, 12 mutations of the X-linked renal specific chloride channel, CLCN5, have been reported in the Dent'sJapan variant. To further identify such CLCN5 mutations and to define the structure-function relationships of this channel, we have investigated five unrelated, non-consanguineous Japanese families with this disorder. Leukocyte DNA from probands was used with CLCN5 primers for PCR amplification of the coding region, and the DNA sequences of the products detd. Functional studies were performed

by expressing the mutants in Xenopus oocytes. Five CLCN5 mutations consisting of two nonsense (R648X and R704X), two missense (S270R and L278F) and one acceptor splice site **mutation** (ag.f[redacted]rw.cg) in intron 4 were identified. The missense and splice site mutations represent novel abnormalities. Heterologous expression in Xenopus oocytes of wild-type and the missense mutants demonstrated that the mutations, which were translated, either abolished or markedly reduced chloride conductance. These results expand the spectrum of CLCN5 mutations assocd. with this renal disorder and provide insight into possible structure-function relationships. For example, both the missense mutations are located within a short putative loop between two transmembrane domains, and our results suggest that this region may have an important functional role in the regulation of channel activity.

ACCESSION NUMBER: 1998:802299 HCAPLUS
DOCUMENT NUMBER: 130:208376
TITLE: Functional characterization of renal chloride channel, CLCN5, mutations associated with Dent'sJapan disease
AUTHOR(S): Igarashi, Takashi; Gunther, Willy; Sekine, Takashi; Inatomi, Jun; Shiraga, Hiroshi; Takahashi, Shouri; Suzuki, Junzou; Tsuru, Noboru; Yanagihara, Toshio; Shimazu, Mitsunobu; Jentsch, Thomas J.; Thakker, Rajesh V.
CORPORATE SOURCE: Department of Pediatrics, Faculty of Medicine, The University of Tokyo, Tokyo, Japan
SOURCE: Kidney Int. (1998), 54(6), 1850-1856
CODEN: KDYIA5; ISSN: 0085-2538
PUBLISHER: Blackwell Science, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 26
REFERENCE(S): (1) Akuta, N; Kidney Int 1997, V52, P911 HCAPLUS
(2) Bolino, A; Eur J Hum Genet 1993, V1, P269 HCAPLUS
(3) Fisher, S; Genomics 1995, V29, P598 HCAPLUS
(4) Fisher, S; Hum Mol Genet 1994, V3, P2053 HCAPLUS
(8) Gunther, W; Proc Natl Acad Sci USA 1998, V95, P8075 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2000 ACS
TI Recurrent T354P **mutation** of the Na+/I symporter in patients with iodide transport defect
AB Iodide transport defect (ITD) is a rare disorder causing congenital hypothyroidism. The authors previously reported that homozygous T354P **mutation** in the sodium/iodide symporter (NIS) gene caused ITD. To clarify the prevalence of this **mutation**, artificial substitution introducing PCR followed by restriction enzyme anal. was developed as a rapid **screening** method to detect the T354P **mutation**. Three apparently unrelated families with ITD, one patient with low thyroid 99mTc pertechnetate (99mTcO4-) uptake and 52 healthy controls (104 alleles) were analyzed for this **mutation**. All families with ITD harbored the **mutation**, suggesting that T354P is a recurrent **mutation** and a major cause of ITD. This was not a widespread **mutation**, because it was not detected in the 52 unrelated normal controls. Because two cases with homozygous T354P **mutation** developed multinodular goiters within their second decade of life though they had been maintained in euthyroid state, homozygous T354P **mutation** alone and/or low intrathyroidal iodide and high serum TSH level in early life might account for tumorigenesis. The patient with low

thyroidal 99mTcO4- uptake did not harbor the T354P **mutation**. Because familial hypocalciuric hypercalcemia was also present in this family, a possibility of the combined abnormality of TSH receptor and calcium functions, which includes an abnormality around the G protein, may be examd. further.

ACCESSION NUMBER: 1998:532073 HCAPLUS
DOCUMENT NUMBER: 129:240482
TITLE: Recurrent T354P **mutation** of the Na+/I symporter in patients with iodide transport defect
AUTHOR(S): Fujiwara, Hirokazu; Tatsumi, Ke-Ita; Miki, Kazunori; Harada, Tokuzo; Okada, Shintaro; Nose, Osamu; Kodama, Soichi; Amino, Nobuyuki
CORPORATE SOURCE: Department of Laboratory Medicine, Osaka University Medical School, Osaka, 565-0871, Japan
SOURCE: J. Clin. Endocrinol. Metab. (1998), 83(8), 2940-2943
CODEN: JCEMAZ; ISSN: 0021-972X
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English

L15 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2000 ACS
TI X-linked hypercalciuric nephrolithiasis: clinical syndromes and chloride channel mutations
AB A review with 82 refs. Topics include: Syndromes of X-linked hypercalciuric nephrolithiasis (X-linked recessive nephrolithiasis, Dent's disease, X-linked recessive hypophosphatemic rickets, low-mol.-wt. proteinuria with **hypercalciuria** and nephrocalcinosis); Common clin. features of the X-linked hypercalciuric nephrolithiasis syndromes (proteinuria, **hypercalciuria**, factors contributing to stone **risk**, other defects of proximal tubular function, defective urinary concn. and acidification, renal failure, rickets/osteomalacia); Positional cloning of the gene CLCN5; CLCN5 mutations in X-linked hypercalciuric nephrolithiasis; Structure of CLC chloride channels and function of CLC-5; Physiol. of CLC-5 and possible pathophysiol. of X-linked hypercalciuric nephrolithiasis; Genetics of idiopathic **hypercalciuria**; and Treatment options in X-linked hypercalciuric nephrolithiasis.

ACCESSION NUMBER: 1998:75395 HCAPLUS
DOCUMENT NUMBER: 128:203593
TITLE: X-linked hypercalciuric nephrolithiasis: clinical syndromes and chloride channel mutations
AUTHOR(S): Scheinman, Steven J.
CORPORATE SOURCE: Department of Medicine, SUNY Health Science Center, Syracuse, NY, USA
SOURCE: Kidney Int. (1998), 53(1), 3-17
CODEN: KDYIA5; ISSN: 0085-2538
PUBLISHER: Blackwell Science, Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

L15 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2000 ACS
TI Mutations of CLCN5 in Japanese children with idiopathic low molecular weight proteinuria, **hypercalciuria** and nephrocalcinosis
AB The annual urinary **screening** of Japanese children above three years of age has identified a progressive renal tubular disorder characterized by low mol. wt. proteinuria, **hypercalciuria** and nephrocalcinosis. The disorder has been obsd. in over 60 patients and has a familial predisposition. Mutations of a renal chloride channel gene, CLCN5, have been reported in four such families, and we have undertaken studies in addnl. patients from 10 unrelated, non-consanguineous Japanese

families to further characterize such CLCN5 mutations and to ascertain their prevalence. CLCN5 abnormalities were identified in 7 of the 10 unrelated patients and consisted of 5 mutations (2 nonsense, 1 frameshift and 2 missense), 1 deletion and 1 silent polymorphism. A clustering of these mutations in CLCN5 exons 8 and 10 was obsd. Over 80% of the CLCN5 mutations could be readily detected by single stranded conformational polymorphism (SSCP) anal., thereby providing a useful **mutation screening** method. Our results, which indicate that over 70% of Japanese patients with this renal tubulopathy have CLCN5 mutations, will help in the genetic and clin. evaluation of children at **risk** from this disorder.

ACCESSION NUMBER: 1997:677523 HCAPLUS
DOCUMENT NUMBER: 127:329894
TITLE: Mutations of CLCN5 in Japanese children with idiopathic low molecular weight proteinuria, **hypercalciuria** and nephrocalcinosis
AUTHOR(S): Akuta, Naoko; Lloyd, Sarah E.; Igarashi, Takashi; Shiraga, Hiroshi; Matsuyama, Takeshi; Yokoro, Seitarou; Cox, Jeremy P. D.; Thakker, Rajesh V.
CORPORATE SOURCE: MRC Molecular Endocrinology Group, MRC Clinical Sciences Centre, Royal Postgraduate Medical School, Hammersmith Hospital, London, UK
SOURCE: Kidney Int. (1997), 52(4), 911-916
CODEN: KDYIA5; ISSN: 0085-2538
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal
LANGUAGE: English

L15 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2000 ACS
TI Idiopathic low molecular weight proteinuria associated with hypercalciuric nephrocalcinosis in Japanese children is due to mutations of the renal chloride channel (CLCN5)
AB The annual urinary **screening** of Japanese children above 3 yr of age has identified a progressive proximal renal tubular disorder characterized by low-mol.-wt. proteinuria, **hypercalciuria**, and nephrocalcinosis. The disorder, which has a familial predisposition and occurs predominantly in males, has similarities to three X-linked proximal renal tubular disorders that are due to mutations in the renal chloride channel gene, CLCN5. Four unrelated Japanese kindreds with this tubulopathy were investigated and 4 different CLCN5 mutations (two nonsense, one missense, and one frameshift) were identified. These are predicted to leads to a loss of chloride channel function, and heterologous expression of the missense CLCN5 **mutation** in Xenopus oocytes demonstrated a 70% redn. in channel activity when compared with the wild-type. In addn., single-stranded conformation polymorphism (SSCP) anal. was a sensitive and specific mutational **screening** method that detected >75% of CLCN5 mutations. Thus, these results expand the spectrum of clin. phenotypes assocd. with CLCN5 mutations to include this proximal renal tubular disorder of Japanese children. In addn., the mutational **screening** of CLCN5 by SSCP will help to supplement the clin. evaluation of the annual urinary **screening** program for this disorder.

ACCESSION NUMBER: 1997:166985 HCAPLUS
DOCUMENT NUMBER: 126:249823
TITLE: Idiopathic low molecular weight proteinuria associated with hypercalciuric nephrocalcinosis in Japanese children is due to mutations of the renal chloride channel (CLCN5)
AUTHOR(S): Lloyd, S. E.; Pearce, S. H. S.; Guenther, W.;

Kawaguchi, H.; Igarashi, T.; Jentsch, T. J.; Thakker, R. V.
CORPORATE SOURCE: MRC Molecular Endocrinology G[redacted], Royal Postgraduate Med. Sch., London, W12, UK
SOURCE: J. Clin. Invest. (1997), 99(5), 967-974
CODEN: JCINAO; ISSN: 0021-9738
PUBLISHER: Rockefeller University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

L15 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2000 ACS
TI Mutations in the Ca2+-sensing receptor gene cause autosomal dominant and sporadic hypoparathyroidism
AB Parathyroid hormone secretion is neg. regulated by a 7-transmembrane domain, G-protein coupled Ca2+-sensing receptor. The authors hypothesized that activating mutations in this receptor might cause autosomal dominant hypoparathyroidism (ADHP). Consistent with this hypothesis, the authors identified, in two families with ADHP, heterozygous missense mutations in the Ca2+-sensing receptor gene that co-segregated with the disorder. None of 50 normal controls had either **mutation**. The authors also identified a de novo, missense Ca2+-sensing receptor **mutation** in a child with severe sporadic hypoparathyroidism. The amino acid substitution in one ADHP family affected the N-terminal, extracellular domain of the receptor. The other mutations involved the transmembrane region. Unlike patients with acquired hypoparathyroidism, patients with these mutations had **hypercalciuria** even at low serum calcium concns. Their greater **hypercalciuria** presumably reflected activation of Ca2+-sensing receptors in kidney cells, where the receptor neg. regulates calcium resorption. This augmented **hypercalciuria** increases the **risk** of renal complications and thus has implications for the choice of therapy.
ACCESSION NUMBER: 1996:287042 HCAPLUS
DOCUMENT NUMBER: 124:339900
TITLE: Mutations in the Ca2+-sensing receptor gene cause autosomal dominant and sporadic hypoparathyroidism
AUTHOR(S): Baron, Jeffrey; Winer, Karen K.; Yanovski, Jack A.; Cunningham, Adrienne W.; Laue, Louisa; Zimmerman, Donald; Cutler, Gordon B., Jr.
CORPORATE SOURCE: Developmental Endocrinology Branch, National Institute Health, Bethesda, MD, 20892-1862, USA
SOURCE: Hum. Mol. Genet. (1996), 5(5), 601-606
CODEN: HMGEE5; ISSN: 0964-6906
DOCUMENT TYPE: Journal
LANGUAGE: English

=> file fsta

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	29.96	81.96

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-5.01	-5.01

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FILE LAST UPDATED: 02 NOV 2000 <20001102/UP>

FILE COVERS 1969 TO DATE.
>>> THE FSTA-THESAURUS IN FIELD /CT HAS BEEN RELOADED <<<

=> s l8

L16 1 HYPERCALCIURIA

=> d l16 ti abs ibib tot

L16 ANSWER 1 OF 1 FSTA COPYRIGHT 2000 IFIS
TI Review of risk factors for osteoporosis with particular reference to a possible aetiological role of dietary salt.
AB Risk factors involved in the aetiology of osteoporosis are reviewed with particular reference to the effects of dietary salt intake on development of this disease. Aspects considered include: definitions and incidence of osteoporosis; conditions associated with osteoporosis; mineral (mainly Ca and P) homeostasis and hormonal regulation of this process; diagnosis, treatment and prevention of osteoporosis; evaluation of various lifestyle, genetic and nutritional risk factors for osteoporosis; effect of dietary Na on urinary Ca excretion (increased Na intake increases urinary Ca concn.); biological significance of **hypercalciuria**; evaluation of evidence from 18 published studies relating to the effects of Na on bone health in humans; Ca absorption and the need for Ca balance studies; and studies, using animal models, of high dietary Na intake. A general discussion of the findings is also presented which concludes that there is no definitive evidence that average daily salt intake (9 g/day) poses a risk for developing osteoporosis.
ACCESSION NUMBER: 2000(08):T0687 FSTA FS FSTA
TITLE: Review of risk factors for osteoporosis with particular reference to a possible aetiological role of dietary salt.
AUTHOR: Cohen, A. J.; Roe, F. J. C.
CORPORATE SOURCE: Toxicol. Advisory Services, Hamilton House, 17 Cedar Rd., Sutton, Surrey SM2 5DA, UK. Tel. (0)20 8642 8265. Fax (0)20 8642 2135
SOURCE: Food and Chemical Toxicology, (2000) 38 (2/3) 237-253,
123 ref.
ISSN: 0278-6915.
DOCUMENT TYPE: General Review (Review)
LANGUAGE: English

=> file uspat

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.20	84.16

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-5.01

FILE 'USPATFULL' ENTERED AT 12:59:53 ON 03 NOV 2000
CA INDEXING COPYRIGHT (C) 2000 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 31 Oct 2000 (20001031/PD)
FILE LAST UPDATED: 31 Oct 2000 (20001031/ED)

HIGHEST PATENT NUMBER: US6141795
CA INDEXING IS CURRENT THROUGH 31 Oct 2000 (20001031/UPCA)
ISSUE CLASS FIELDS (/[redacted]) CURRENT THROUGH: 31 Oct 200[redacted]20001031/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Jul 2000
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Jul 2000

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>>> Image data for the /FA field are available the following week. <<<

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>>> USPTO Manual of Classifications in the /NCL, /INCL, and /RPCL <<<
>>> fields. This thesaurus includes catchword terms from the <<<
>>> USPTO/MOC subject headings and subheadings. Thesauri are also <<<
>>> available for the WIPO International Patent Classification <<<
>>> (IPC) Manuals, editions 1-6, in the /IC1, /IC2, /IC3, /IC4, <<<
>>> /IC5, and /IC (/IC6) fields, respectively. The thesauri in <<<
>>> the /IC5 and /IC fields include the corresponding catchword <<<
>>> terms from the IPC subject headings and subheadings. <<<

This file contains CAS Registry Numbers for easy and accurate
substance identification.

=> s l8

L17 104 HYPERCALCIURIA

=> s l17 and mutation

14340 MUTATION
L18 8 L17 AND MUTATION

=> s l18 and (screening?)

64216 SCREENING?
L19 7 L18 AND (SCREENING?)

=> d l19 ti abs ibib tot

L19 ANSWER 1 OF 7 USPATFULL
TI Treatment of balance impairments
AB Compositions and methods are provided for prophylactic or therapeutic
treatment of balance impairments involving neuronal damage, loss, or
degeneration, preferably of vestibular ganglion neurons, in an animal
by
administration of an effective amount of a trkB or trkC agonist,
particularly a neurotrophin, more preferably NT-4/5.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2000:125012 USPATFULL
TITLE: Treatment of balance impairments
INVENTOR(S): Gao, Wei-Qiang, Foster City, CA, United States
PATENT ASSIGNEE(S): Genentech, Inc., South San Francisco, CA, United
States
(U.S. corporation)

	NUMBER	DATE	
PATENT INFORMATION:	US 6121235	20000919	
APPLICATION INFO.:	US 1995-581662	19951229	(8)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Jones, Dwayne C.
ASSISTANT EXAMINER: Delacroix-Muirheid, C.
LEGAL REPRESENTATIVE: Knobbe, Martens, Olson & Bear LLP
NUMBER OF CLAIMS: 49
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 3419
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 2 OF 7 USPATFULL
TI Calcium receptor-active molecules
AB The present invention relates to the different roles inorganic ion receptors have in cellular and body processes. The present invention features: (1) molecules which can modulate one or more inorganic ion receptor activities, preferably the molecule can mimic or block an effect of an extracellular ion on a cell having an inorganic ion receptor, more preferably the extracellular ion is Ca.sup.2+ and the effect is on a cell having a calcium receptor; (2) inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (3) nucleic acids encoding inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (4) antibodies and fragments thereof, targeted to inorganic ion receptor proteins, preferably calcium receptor protein; and (5) uses of such molecules, proteins, nucleic acids and antibodies.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2000:24677 USPATFULL
TITLE: Calcium receptor-active molecules
INVENTOR(S): Nemeth, Edward F., Salt Lake City, UT, United States
Van Wagenen, Bradford C., Salt Lake City, UT, United States
Balandrin, Manuel F., Sandy, UT, United States
DelMar, Eric G., Salt Lake City, UT, United States
Moe, Scott T., Salt Lake City, UT, United States
PATENT ASSIGNEE(S): NPS Pharmaceuticals, Inc., Salt Lake City, UT, United States (U.S. corporation)
The Brigham and Women's Hospital, Boston, MA, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6031003	20000229
APPLICATION INFO.:	US 1995-484719	19950607 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-353784, filed on 8 Dec 1994 which is a continuation-in-part of Ser. No. WO 1994-US12117, filed on 21 Oct 1994 Ser. No. Ser. No. US 1994-292827, filed on 19 Aug 1994, now abandoned Ser. No. Ser. No. US 1993-141248, filed on 22 Oct 1993, now abandoned And Ser. No. US 1993-9389, filed on 23 Feb 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-17127, filed on 12 Feb 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-934161, filed on 21 Aug 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-834044, filed on 11 Feb 1992, now abandoned which

is a continuation-in-part of Ser. No. US 1991-749451, filed on 23 Aug 1991, now abandoned
DOCUMENT TYPE: tility
PRIMARY EXAMINER: Tsang, Cecilia J.
ASSISTANT EXAMINER: Borin, Michael
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 145
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 109 Drawing Figure(s); 85 Drawing Page(s)
LINE COUNT: 8955
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 3 OF 7 USPATFULL
TI Calcium receptor-active molecules
AB The present invention relates to the different roles inorganic ion receptors have in cellular and body processes. The present invention features: (1) molecules which can modulate one or more inorganic ion receptor activities, preferably the molecule can mimic or block an effect of an extracellular ion on a cell having an inorganic ion receptor, more preferably the extracellular ion is Ca.sup.2+ and the effect is on a cell having a calcium receptor; (2) inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (3) nucleic acids encoding inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (4) antibodies and fragments thereof, targeted to inorganic ion receptor proteins, preferably calcium receptor protein; and (5) uses of such molecules, proteins, nucleic acids and antibodies.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2000:1911 USPATFULL
TITLE: Calcium receptor-active molecules
INVENTOR(S): Nemeth, Edward F., Salt Lake City, UT, United States
Van Wagenen, Bradford C., Salt Lake City, UT, United States
Balandrin, Manuel F., Sandy, UT, United States
DelMar, Eric G., Salt Lake City, UT, United States
Moe, Scott T., Salt Lake City, UT, United States
PATENT ASSIGNEE(S): NPS Pharmaceuticals, Inc., Salt Lake City, UT, United States (U.S. corporation)
The Brigham and Women's Hospital, Boston, MA, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6011068	20000104
APPLICATION INFO.:	US 1994-353784	19941208 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 1994-US12117, filed on 21 Oct 1994 And a continuation-in-part of Ser. No. US 1994-292827, filed on 19 Aug 1994, now abandoned And a continuation-in-part of Ser. No. US 1993-141248, filed on 22 Oct 1993, now abandoned And a continuation-in-part of Ser. No. US 1993-9389, filed on 23 Feb 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-17127, filed on 12 Feb 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-934161, filed on 21 Aug 1992, now abandoned which is a

continuation-in-part of Ser. No. US 1992-834044, filed on 11 Feb 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-749451, filed on 23 Aug 1991, now abandoned
DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Henley, III, Raymond
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 103
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 111 Drawing Figure(s); 85 Drawing Page(s)
LINE COUNT: 7466
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 4 OF 7 USPATFULL
TI Calcium receptor-active molecules
AB The present invention relates to the different roles inorganic ion receptors have in cellular and body processes. The present invention features: (1) molecules which can modulate one or more inorganic ion receptor activities, preferably the molecule can mimic or block an effect of an extracellular ion on a cell having an inorganic ion receptor, more preferably the extracellular ion is Ca.sup.2+ and the effect is on a cell having a calcium receptor; (2) inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (3) nucleic acids encoding inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (4) antibodies and fragments thereof, targeted to inorganic ion receptor proteins, preferably calcium receptor protein; and (5) uses of such molecules, proteins, nucleic acids and antibodies.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 1999:121216 USPATFULL
TITLE: Calcium receptor-active molecules
INVENTOR(S): Brown, Edward M., Milton, MA, United States
Hebert, Steven C., Wellesley, MA, United States
Garrett, Jr., James E., Salt Lake City, UT, United States
PATENT ASSIGNEE(S): NPS Pharmaceuticals, Inc., Salt Lake City, UT, United States (U.S. corporation)
Brigham and Women's Hospital, Boston, MA, United States
(U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5962314	19991005
APPLICATION INFO.:	US 1997-943986	19971003 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-484565, filed on 7 Jun 1995, now patented, Pat. No. US 5763569 which is a continuation-in-part of Ser. No. US 1994-353784, filed on 8 Dec 1994 which is a continuation-in-part of Ser. No. WO 1994-US12117, filed on 21 Oct 1994 Ser. No. Ser. No. US 1994-292827, filed on 19 Aug 1994, now abandoned
Ser. No. Ser. No. US 1993-141248, filed on 22 Oct 1993, now abandoned And Ser. No. US 1993-9389, filed on 23 Feb 1993, now abandoned
DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Ulm, John
ASSISTANT EXAMINER: Saoud, Christine
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 111 Drawing Figure(s); 85 Drawing Page(s)
LINE COUNT: 7882
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 5 OF 7 USPATFULL
TI Method of **screening** calcium receptor-active molecules
AB The present invention relates to the different roles inorganic ion receptors have in cellular and body processes. The present invention features: (1) molecules which can modulate one or more inorganic ion receptor activities, preferably the molecule can mimic or block an effect of an extracellular ion on a cell having an inorganic ion receptor, more preferably the extracellular ion is Ca.sup.2+ and the effect is on a cell having a calcium receptor; (2) inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (3) nucleic acids encoding inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (4) antibodies and fragments thereof, targeted to inorganic ion receptor proteins, preferably calcium receptor protein; and (5) uses of such molecules, proteins, nucleic acids and antibodies.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 1999:4350 USPATFULL
TITLE: Method of **screening** calcium receptor-active molecules
INVENTOR(S): Nemeth, Edward F., Salt Lake City, UT, United States
Brown, Edward M., Milton, MA, United States
Hebert, Steven C., Wellesley, MA, United States
Garrett, Jr., James E., Salt Lake City, UT, United States
Van Wagenen, Bradford C., Salt Lake City, UT, United States
Balandrin, Manuel F., Sandy, UT, United States
Del Mar, Eric G., Salt Lake City, UT, United States
PATENT ASSIGNEE(S): The Brigham and Women's Hospital, Inc., Boston, MA, United States (U.S. corporation)
NPS Pharmaceuticals, Inc., Salt Lake City, UT, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5858684	19990112
APPLICATION INFO.:	US 1995-480751	19950607 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-353784, filed on 8 Dec 1994 which is a continuation-in-part of Ser. No. US 1994-292827, filed on 19 Aug 1994, now abandoned
And a continuation-in-part of Ser. No. US 1993-141248, filed on 22 Oct 1993, now abandoned And a continuation-in-part of Ser. No. US 1993-9389, filed on 23 Feb 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-17127, filed on 12 Feb 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-934161, filed on 21 Aug 1992, now abandoned which is a

continuation-in-part of Ser. No. US 1992-834044, filed on 11 Feb 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-749451, filed on 23 Aug 1991, now abandoned

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Walsh, Stephen
ASSISTANT EXAMINER: Sorensen, Kenneth A.
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 48
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 111 Drawing Figure(s); 85 Drawing Page(s)
LINE COUNT: 7588
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 6 OF 7 USPATFULL
TI Calcium receptor-active molecules
AB The present invention features calcium receptor polypeptides and fragments thereof. Uses of a calcium receptor polypeptide include providing a polypeptide having the activity of a calcium receptor polypeptide. Calcium receptor polypeptide fragments can be used, for example, to generate antibodies to a calcium receptor polypeptide.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 1998:65348 USPATFULL
TITLE: Calcium receptor-active molecules
INVENTOR(S): Brown, Edward M., Milton, MA, United States
Hebert, Steven C., Wellesley, MA, United States
Garrett, Jr., James E., Salt Lake City, UT, United States
PATENT ASSIGNEE(S): The Brigham and Women's Hospital, Inc, Boston, MA, United States (U.S. corporation)
NPS Pharmaceuticals, Inc., Salt Lake City, UT, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5763569	19980609
APPLICATION INFO.:	US 1995-484565	19950607 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-353784, filed on 8 Dec 1994 which is a continuation-in-part of Ser. No. US 1994-292827, filed on 19 Aug 1994, now abandoned
Ser. No. Ser. No. US 1993-141248, filed on 22 Oct 1993,
now abandoned And Ser. No. US 1993-9389, filed on 23 Feb 1993, now abandoned , said Ser. No. US -292827 which is a continuation-in-part of Ser. No. US -141248 which is a continuation-in-part of Ser. No. US -9389 And a continuation-in-part of Ser. No. US 1993-17127, filed on 12 Feb 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-934161, filed on 21 Aug 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-834044, filed on 11 Feb 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-749451, filed on 23 Aug 1991, now abandoned
DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Walsh, Stephen
ASSISTANT EXAMINER: Sorensen, Kenneth A.
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 13
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 111 Drawing Figure(s); 85 Drawing Page(s)

LINE COUNT: 6942
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 7 OF 7 USPATFULL
TI Calcium receptor-active molecules
AB The present invention relates to the different roles inorganic ion receptors have in cellular and body processes. The present invention features: (1) molecules which can modulate one or more inorganic ion receptor activities, preferably the molecule can mimic or block an effect of an extracellular ion on a cell having an inorganic ion receptor, more preferably the extracellular ion is Ca.sup.2+ and the effect is on a cell having a calcium receptor; (2) inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (3) nucleic acids encoding inorganic ion receptor proteins and fragments thereof, preferably calcium receptor proteins and fragments thereof; (4) antibodies and fragments thereof, targeted to inorganic ion receptor proteins, preferably calcium receptor protein; and (5) uses of such molecules, proteins, nucleic acids and antibodies.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	97:107219 USPATFULL
TITLE:	Calcium receptor-active molecules
INVENTOR(S):	Brown, Edward M., Milton, MA, United States Fuller, Forrest H., Salt Lake City, UT, United States Hebert, Steven C., Wellesley, MA, United States Garrett, Jr., James E., Salt Lake City, UT, United States
PATENT ASSIGNEE(S):	The Brigham & Women's Hospital, Inc., Boston, MA, United States (U.S. corporation) NPS Pharmaceuticals, Inc., Salt Lake City, UT, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5688938	19971118
APPLICATION INFO.:	US 1995-485588	19950607 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-353784, filed on 8 Dec 1994 which is a continuation-in-part of Ser. No. US 1993-9389, filed on 23 Feb 1993, now abandoned Ser. No. Ser. No. US 1993-141248, filed on 22 Oct 1993, now abandoned And Ser. No. US 1994-292827, filed on 19 Aug 1994, now abandoned which is a continuation-in-part of Ser. No. US -141248 which is a continuation-in-part of Ser. No. US -9389 which is a continuation-in-part of Ser. No. US 1993-17127, filed on 12 Feb 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-934161, filed on 21 Aug 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-834044, filed on 11 Feb 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-749451, filed on 23 Aug 1991, now abandoned

DOCUMENT TYPE:	Utility
PRIMARY EXAMINER:	Walsh, Stephen
ASSISTANT EXAMINER:	Sorensen, Kenneth A.
LEGAL REPRESENTATIVE:	Lyons & Lyons LLP
NUMBER OF CLAIMS:	24
EXEMPLARY CLAIM:	1

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NUMBER OF DRAWINGS:      111 Drawing Figure(s); 84 Drawing Page(s)
LINE COUNT:              [redacted]522
CAS INDEXING IS AVAILAB[redacted] FOR THIS PATENT.

=> d his

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     FILE 'HCAOLD' ENTERED AT 12:41:54 ON 03 NOV 2000
               E REED-GITOMER,B/AU
               E PAK, C/AU

     FILE 'REGISTRY' ENTERED AT 12:45:48 ON 03 NOV 2000
               E HYPERCALCIURIA
L1             2 S E3

     FILE 'HCAOLD' ENTERED AT 12:46:53 ON 03 NOV 2000
L2            19 S HYPERCALCIURIA
L3             0 S L1 AND L2
L4             0 S L2 AND SCREENING
L5             0 S L2 AND (DETECTION METHOD OR SCREENING OR TESTING OR RISK
ASSE
L6             0 S L2 AND (RISK OR INCREASED RISK OR RISK ASSESSMENT?)

     FILE 'BIOSIS' ENTERED AT 12:51:33 ON 03 NOV 2000
L7             0 S L1
L8          1896 S HYPERCALCIURIA
L9           177 S L8 AND (METHOD OF SCREENING OR RISK OR RISK ASSESSMENT OR
DET
L10            5 S L9 AND (INCREASED RISK?)
L11            1 S L8 AND GENETIC MUTATION

     FILE 'HCAPLUS' ENTERED AT 12:55:37 ON 03 NOV 2000
L12          662 S L8
L13           44 S L12 AND MUTATION
L14            1 S L13 AND CHROMOSOME 1
L15            8 S L13 AND (SCREENING OR RISK OR INCREASE RISK OR RISK
ASSESSMEN

     FILE 'FSTA' ENTERED AT 12:59:26 ON 03 NOV 2000
L16            1 S L8

     FILE 'USPATFULL' ENTERED AT 12:59:53 ON 03 NOV 2000
L17          104 S L8
L18            8 S L17 AND MUTATION
L19            7 S L18 AND (SCREENING?)

=> file frosti

COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                 ENTRY    SESSION
FULL ESTIMATED COST                              12.74      96.90

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)    SINCE FILE      TOTAL
                                                 ENTRY    SESSION
CA SUBSCRIBER PRICE                               0.00      -5.01

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FILE LAST UPDATED: 18 OCT 2000      <20001018/UP>
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=> s l8

L20         17 HYPERCALCIURIA

=> s l20 and mutation

           348 MUTATION
L21          0 L20 AND MUTATION

=> s l20 and (risk or risk assessment or screening or detection?)

          9881 RISK
          9881 RISK
          5269 ASSESSMENT
           905 RISK ASSESSMENT
                 (RISK(W)ASSESSMENT)
          2529 SCREENING
         24054 DETECTION?
L22          3 L20 AND (RISK OR RISK ASSESSMENT OR SCREENING OR DETECTION?)

=> d l22 ti abs ibib tot


L22   ANSWER 1 OF 3  FROSTI  COPYRIGHT 2000 LFRA
TI    Review of risk factors for osteoporosis with particular
      reference to a possible aetiological role of dietary salt.
AN    522383   FROSTI
AB    The published literature was reviewed to establish whether excessive
salt
      intake was an important risk factor for the development of
      osteoporosis and whether an intervention strategy based on salt
      restriction would be beneficial in the prevention of osteoporosis.
      Genetic factors appear to be far more important than nutritional,
      hormonal, environmental and lifestyle factors in the pathogenesis of
      osteoporosis. Preventive measures should be aimed at maximizing bone
mass
      at skeletal maturity and retarding bone loss thereafter, possibly by
      increased intakes of potassium, magnesium, zinc, vitamin C, fibre and
      alkaline-producing fruit and vegetables. High salt intakes may lead to
      higher urinary calcium output in a minority of humans, but calcium
      homeostasis is normally well regulated so that increased sodium intake
      neither exerts a consistent effect on bone health nor leads to
      irreversible changes in bone modelling. The authors conclude that there
      is no sound evidence that consumption of salt at the present average
      level of 9 g/day constitutes a risk to osteoporosis for the
      generally healthy person, and that a reduction of salt intake to 6 g/day
      would not be beneficial as an intervention measure in the prevention of
      osteoporosis (although excessively high levels of dietary salt should be
      avoided by persons with raised blood pressure or a limited range of
      genetic disorders). More research is recommended to assess the long-term
      effects of various nutrients (including sodium) on bone health at
current
      and reduced intake levels, and to determine whether subpopulations exist
      in which adaptation to sodium-induced hypercalciuria may be
      compromised.
TITLE:                  Review of risk factors for osteoporosis with
                        particular reference to a possible aetiological role
                        of dietary salt.
AUTHOR:                 Cohen A.J.; Ro F.J.C.
SOURCE:                 Food and Chemical Toxicology, 2000, (February-March),
                        38 (2-3), 237-253 (123 ref.)
                        ISSN: 0278-6915
```

DOCUMENT TYPE: Journal
LANGUAGE: English
SUMMARY LANGUAGE: English

L22 ANSWER 2 OF 3 FROSTI COPYRIGHT 2000 LFRA
TI Dietary salt, urinary calcium, and kidney stone **risk**.
AN 383938 FROSTI
AB This brief review discusses the recent re-evaluation of the role that a reduced-salt diet may have in the pathogenesis of calcium renal stones. Both salt-loading studies and reports of free-living populations find that an increase of 2300 mg of dietary sodium results in an increase in urinary excretion of approximately 40 mg of calcium. Patients with **hypercalciuria** may benefit from a reduction in salt intake to no more than 100 mmol/day, but it is still not clear whether a low-salt diet can prevent calcium stone formation.
TITLE: Dietary salt, urinary calcium, and kidney stone **risk**.
AUTHOR: Massey L.K.; Whiting S.J.
SOURCE: Nutrition Reviews, 1995, 53 (5), 131-134 (31 ref.)
DOCUMENT TYPE: Journal
LANGUAGE: English
SUMMARY LANGUAGE: English

L22 ANSWER 3 OF 3 FROSTI COPYRIGHT 2000 LFRA
TI Dietary **hypercalciuria** in patients with calcium oxalate kidney stones.
AN 356265 FROSTI
AB Kidney stones (nephrolithiasis), composed of calcium oxalate, affect a significant number of the American population. **Hypercalciuria** (high excretion of calcium in the urine), is known to be a **risk** factor for developing kidney stones. This work studied patients with calcium oxalate kidney stones to investigate the effect of diet (such as sodium, calcium and phosphorus intakes) on **hypercalciuria** and increasing the **risk** of kidney stones. Of the 282 patients with kidney stones, 124 were found to be hypercalciuric on their usual diet or on a high calcium-defined diet. Of the hypercalciuric patients, many excreted more calcium on the free diet than on the calcium diet, suggesting that part of their usual diet was contributing to the **hypercalciuria**. Further analysis suggested that dietary sodium, calcium, phosphorus and, to a lesser extent, carbohydrate and protein contributed to calcium excretion. The authors concluded that diet, especially a high sodium intake, might contribute to **hypercalciuria** in patients with kidney stones.
TITLE: Dietary **hypercalciuria** in patients with calcium oxalate kidney stones.
AUTHOR: Burtis W.J.; Gay L.; Insogna K.L.; Ellison A.; Broadus A.E.
SOURCE: American Journal of Clinical Nutrition, 1994, 60 (3), 424-429 (31 ref.)
DOCUMENT TYPE: Journal
LANGUAGE: English
SUMMARY LANGUAGE: English

=> d his

(FILE 'HOME' ENTERED AT 12:41:35 ON 03 NOV 2000)

FILE 'HCAOLD' ENTERED AT 12:41:54 ON 03 NOV 2000

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                E REED-GITOMER,B/AU
                E PAK, C/AU

     FILE 'REGISTRY' ENTERED AT 12:45:48 ON 03 NOV 2000
                E HYPERCALCIURIA
L1             2 S E3

     FILE 'HCAOLD' ENTERED AT 12:46:53 ON 03 NOV 2000
L2            19 S HYPERCALCIURIA
L3             0 S L1 AND L2
L4             0 S L2 AND SCREENING
L5             0 S L2 AND (DETECTION METHOD OR SCREENING OR TESTING OR RISK
ASSE
L6             0 S L2 AND (RISK OR INCREASED RISK OR RISK ASSESSMENT?)

     FILE 'BIOSIS' ENTERED AT 12:51:33 ON 03 NOV 2000
L7             0 S L1
L8          1896 S HYPERCALCIURIA
L9           177 S L8 AND (METHOD OF SCREENING OR RISK OR RISK ASSESSMENT OR
DET
L10            5 S L9 AND (INCREASED RISK?)
L11            1 S L8 AND GENETIC MUTATION

     FILE 'HCAPLUS' ENTERED AT 12:55:37 ON 03 NOV 2000
L12          662 S L8
L13           44 S L12 AND MUTATION
L14            1 S L13 AND CHROMOSOME 1
L15            8 S L13 AND (SCREENING OR RISK OR INCREASE RISK OR RISK
ASSESSMEN

     FILE 'FSTA' ENTERED AT 12:59:26 ON 03 NOV 2000
L16            1 S L8

     FILE 'USPATFULL' ENTERED AT 12:59:53 ON 03 NOV 2000
L17          104 S L8
L18            8 S L17 AND MUTATION
L19            7 S L18 AND (SCREENING?)

     FILE 'FROSTI' ENTERED AT 13:01:19 ON 03 NOV 2000
L20           17 S L8
L21            0 S L20 AND MUTATION
L22            3 S L20 AND (RISK OR RISK ASSESSMENT OR SCREENING OR DETECTION?)

=> s l20 and (disease risk or increased risk or risk factor?)

         13554 DISEASE
          9881 RISK
           235 DISEASE RISK
                 (DISEASE(W)RISK)
         18538 INCREASED
          9881 RISK
           572 INCREASED RISK
                 (INCREASED(W)RISK)
          9881 RISK
         44433 FACTOR?
          1964 RISK FACTOR?
                 (RISK(W)FACTOR?)
L23            2 L20 AND (DISEASE RISK OR INCREASED RISK OR RISK FACTOR?)

=> d l23 ti abs ibib tot


L23   ANSWER 1 OF 2  FROSTI  COPYRIGHT 2000 LFRA
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TI Review of **risk factors** for osteoporosis with particular reference to a possible aetiological role of dietary salt.
AN 522383 FROSTI
AB The published literature was reviewed to establish whether excessive salt intake was an important **risk factor** for the development of osteoporosis and whether an intervention strategy based on salt restriction would be beneficial in the prevention of osteoporosis. Genetic factors appear to be far more important than nutritional, hormonal, environmental and lifestyle factors in the pathogenesis of osteoporosis. Preventive measures should be aimed at maximizing bone mass at skeletal maturity and retarding bone loss thereafter, possibly by increased intakes of potassium, magnesium, zinc, vitamin C, fibre and alkaline-producing fruit and vegetables. High salt intakes may lead to higher urinary calcium output in a minority of humans, but calcium homeostasis is normally well regulated so that increased sodium intake neither exerts a consistent effect on bone health nor leads to irreversible changes in bone modelling. The authors conclude that there is no sound evidence that consumption of salt at the present average level of 9 g/day constitutes a risk to osteoporosis for the generally healthy person, and that a reduction of salt intake to 6 g/day would not be beneficial as an intervention measure in the prevention of osteoporosis (although excessively high levels of dietary salt should be avoided by persons with raised blood pressure or a limited range of genetic disorders). More research is recommended to assess the long-term effects of various nutrients (including sodium) on bone health at current and reduced intake levels, and to determine whether subpopulations exist in which adaptation to sodium-induced **hypercalciuria** may be compromised.

TITLE:	Review of **risk factors** for osteoporosis with particular reference to a possible aetiological role of dietary salt.
AUTHOR:	Cohen A.J.; Ro F.J.C.
SOURCE:	Food and Chemical Toxicology, 2000, (February-March), 38 (2-3), 237-253 (123 ref.) ISSN: 0278-6915
DOCUMENT TYPE:	Journal
LANGUAGE:	English
SUMMARY LANGUAGE:	English

L23 ANSWER 2 OF 2 FROSTI COPYRIGHT 2000 LFRA
TI Dietary **hypercalciuria** in patients with calcium oxalate kidney stones.
AN 356265 FROSTI
AB Kidney stones (nephrolithiasis), composed of calcium oxalate, affect a significant number of the American population. **Hypercalciuria** (high excretion of calcium in the urine), is known to be a **risk factor** for developing kidney stones. This work studied patients with calcium oxalate kidney stones to investigate the effect of diet (such as sodium, calcium and phosphorus intakes) on **hypercalciuria** and increasing the risk of kidney stones. Of the 282 patients with kidney stones, 124 were found to be hypercalciuric on their usual diet or on a high calcium-defined diet. Of the hypercalciuric patients, many excreted more calcium on the free diet than on the calcium diet, suggesting that part of their usual diet was contributing to the **hypercalciuria**. Further analysis suggested that dietary sodium, calcium, phosphorus and, to a lesser extent, carbohydrate and protein contributed to calcium excretion. The authors concluded that diet,

```
      especially a high sodium intake, might contribute to
    hypercalciuria in patients with kidney stones.
TITLE:                  Dietary hypercalciuria in patients with
                        calcium oxalate kidney stones.
AUTHOR:                 Burtis W.J.; Gay L.; Insogna K.L.; Ellison A.;
Broadus
                        A.E.
SOURCE:                 American Journal of Clinical Nutrition, 1994, 60 (3),
                        424-429 (31 ref.)
DOCUMENT TYPE:          Journal
LANGUAGE:               English
SUMMARY LANGUAGE:       English


=> file wpids

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     8.55     105.45

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                                                       ENTRY    SESSION
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DERWENT WEEK FOR POLYMER INDEXING:  200055
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

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=> s l8

L24         36 HYPERCALCIURIA

=> s l24 and (risk or risk assessment or screening or detection?)

         51554 RISK
         51554 RISK
          5180 ASSESSMENT
            52 RISK ASSESSMENT
                 (RISK(W)ASSESSMENT)
         26121 SCREENING
        205404 DETECTION?
L25          3 L24 AND (RISK OR RISK ASSESSMENT OR SCREENING OR DETECTION?)

=> d l25 ti abs ibib tot


L25  ANSWER 1 OF 3  WPIDS COPYRIGHT 2000   DERWENT INFORMATION LTD
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TI Novel genomic region useful in **screening** for absorptive **hypercalciuria** or osteoporosis with **hypercalciuria**.

AN 2000-116959 [10] WPIDS

AB WO 9967426 A UPAB: 20000228

NOVELTY - A method for **screening** for an increased **risk** of **hypercalciuria**, comprising obtaining a sample nucleic acid from a subject, and analyzing it to detect the presence or absence of a genetic mutation in genomic region associated with an increased **risk** of developing **hypercalciuria**, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a method of treating **hypercalciuria**, comprising **screening** for an increased **risk** of **hypercalciuria** using a sample nucleic acid from a patient, detecting the increased **risk** of **hypercalciuria** and treating the patient with an increased **risk** of **hypercalciuria**; and

(2) a method for familial **screening** for an increased **risk** of absorptive **hypercalciuria** or osteoporosis with **hypercalciuria**, comprising obtaining a sample nucleic acid from a patient, and analyzing the sample nucleic acid to detect the presence of a marker known to be linked genetically to a region of human chromosome1, where the region is associated with an increased **risk** of developing absorptive **hypercalciuria**.

ACTIVITY - Osteopathic; anticalciuric;

MECHANISM OF ACTION - None given

USE - Identification of the genomic region allows **screening** for an increased **risk** of developing absorptive **hypercalciuria** or osteoporosis with **hypercalciuria**, by analyzing a nucleic acid sample from a subject (claimed) and detecting a genetic mutation in the region (claimed). Absorptive **hypercalciuria** causes stone formation in approximately half of reported cases of the common clinical disorder nephrolithiasis, and is frequently accompanied by osteoporosis with **hypercalciuria**; the region is therefore useful to screen for osteoporosis with **hypercalciuria**, including ideopathic osteoporosis with **hypercalciuria** and postmenopausal osteoporosis with **hypercalciuria** (claimed). Such **screening** allows patients at **risk** of the above conditions to identified and treated (claimed), e.g. by known therapies or dietary or fluid regimes, optionally combined with treatment to prevent stone formation (claimed). Familial **screening** for an increased **risk** of absorptive **hypercalciuria** or osteoporosis with **hypercalciuria** may also be possible using the identified region, by analyzing nucleic acid samples for a marker known to be linked genetically to the region (claimed). Identification of the region allows polynucleotides and encoded polypeptides associated with absorptive **hypercalciuria** or osteoporosis with **hypercalciuria** to be identified, useful e.g. therapeutically to treat conditions as above, diagnostically and to identify polypeptide modulators etc.

ADVANTAGE - Identification of the region allows a straightforward genetic test to be used to diagnose absorptive **hypercalciuria** and/or osteoporosis with **hypercalciuria**, eliminating the complications of extended blood and urine testing and patient compliance with a defined diet necessary in previous **screening** methods.

Dwg.0/2

ACCESSION NUMBER: 2000-116959 [10] WPIDS

DOC. NO. CPI: C2000-035816

TITLE: Novel genomic region useful in **screening** for absorptive **hypercalciuria** or osteoporosis with

hypercalciuria.

DERWENT CLASS: B04 D16
INVENTOR(S): [redacted], C Y C; REED-GITOMER, B Y
PATENT ASSIGNEE(S): (TEXA) UNIV TEXAS SYSTEM
COUNTRY COUNT: 86
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9967426	A1	19991229	(200010)*	EN	153

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW

AU 9948310	A	20000110	(200025)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9967426	A1	WO 1999-US14347	19990623
AU 9948310	A	AU 1999-48310	19990623

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9948310	A	Based on	WO 9967426

PRIORITY APPLN. INFO: US 1998-90348 19980623

L25 ANSWER 2 OF 3 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
TI Inhibiting hyper-proliferative prostatic cancer or hyperplasia - and promoting cell differentiation by administering hydroxy vitamin D derivatives, optionally with another anticancer agent e.g. cisplatin.
AN 1998-347379 [30] WPIDS
CR 1990-083366 [11]; 1992-132038 [16]; 1996-476702 [47]; 1997-051865 [05]; 1997-051866 [05]; 1999-152844 [13]
AB US 5763429 A UPAB: 20001001
Inhibition of hyperproliferative activity of human prostatic neoplastic or hyperplastic cells comprises treating the cells with a 1 alpha -hydroxyvitamin D compound (I) having a hydrocarbon moiety substituted at C-24.

USE - (I) are used for the treatment of prostatic cancer or hyperplasia by inhibiting hyperproliferative activity and enhancing differentiation in prostatic cells. (I) decrease or stabilise the cellular abnormal proliferative activity of the cancer (all claimed).

ADVANTAGE - (I) or their in vivo metabolites have a vitamin D receptor binding affinity and promotion of cell differentiation equivalent to that of 1- alpha ,25-dihydroxyvitamin D3 (II) but the **risk** of inducing dose-limiting hypercalcaemia and **hypercalciuria** is reduced.

Dwg.0/0

ACCESSION NUMBER: 1998-347379 [30] WPIDS
CROSS REFERENCE: 1990-083366 [11]; 1992-132038 [16]; 1996-476702 [47]; 1997-051865 [05]; 1997-051866 [05]; 1999-152844 [13]
DOC. NO. CPI: C1998-107338
TITLE: Inhibiting hyper-proliferative prostatic cancer or

hyperplasia - and promoting cell differentiation by administering hydroxy vitamin D derivatives, optionally with another anticancer agent e.g. cisplatin.

DERWENT CLASS: B05
INVENTOR(S): BISHOP, C W; KNUTSON, J C; MAZESS, R B
PATENT ASSIGNEE(S): (BONE-N) BONE CARE INT INC
COUNTRY COUNT: 69
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 5763429	A	19980609	(199830)*		10
WO 9829123	A1	19980709	(199833)	EN	
RW: AT BE CH DE DK ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LT LU LV MD MG MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TT UA UG UZ VN					
AU 9855956	A	19980731	(199849)		
EP 948334	A1	19991013	(199947)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
BR 9713663	A	20000404	(200030)		
CN 1248917	A	20000329	(200033)		
CZ 9902355	A3	20000614	(200037)		
AU 723835	B	20000907	(200048)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 5763429	A	CIP of	US 1993-119895	19930910
		CIP of	US 1994-265438	19940624
		CIP of	US 1995-415488	19950403
		CIP of	US 1995-486387	19950607
			US 1996-781910	19961230
WO 9829123	A1		WO 1997-US22450	19971210
AU 9855956	A		AU 1998-55956	19971210
EP 948334	A1		EP 1997-952316	19971210
			WO 1997-US22450	19971210
BR 9713663	A		BR 1997-13663	19971210
			WO 1997-US22450	19971210
CN 1248917	A		CN 1997-181974	19971210
CZ 9902355	A3		WO 1997-US22450	19971210
			CZ 1999-2355	19971210
AU 723835	B		AU 1998-55956	19971210

FILING DETAILS:

PATENT NO	KIND		PATENT NO
US 5763429	A	CIP of	US 5403831
		CIP of	US 5602116
		CIP of	US 5674859
AU 9855956	A	Based on	WO 9829123
EP 948334	A1	Based on	WO 9829123
BR 9713663	A	Based on	WO 9829123
CZ 9902355	A3	Based on	WO 9829123
AU 723835	B	Previous Publ.	AU 9855956
		Based on	WO 9829123

PRIORITY APPLN. INFO: US 1996-781910 19961230; US 1993-119895 19930910; US 1994-265438 19940624; US 1995-415488 19950403; US 1995-486387 19950607

L25 ANSWER 3 OF 3 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
TI Topical compsn. f[redacted] disorders due to decreased cal[redacted]m transport - contg. vitamin-D2 or -D3 active cpds. or provitamin(s).
AN 1980-82501C [46] WPIDS
AB US 4230701 A UPAB: 19930902
Pharmaceutical compsn. comprises a topical carrier together with a vitamin D2 or D3 active material of formula (I) or a provitamin of formula (II) or (III). In the formulae Z is CHMe-CHA-CHB-CHR2-CMeR4-CH2R3; R is CH2= or Me; R2 is H, OH or Me; R1R3 and R4 are H, OH or halogen; and A and B are H or A+B form a double bond; provided that >=1 of R1-R4 is OH, and when A+B form a double bond, R2 is Me.

Compsns. are useful for treating disorders due to decreased Ca transport. The skin acts as a transport medium for (I)-(III), so that they enter the blood stream in a slow and controlled manner in contrast to oral or parenteral admin. One topical appln. can last for >=2 weeks and **risk** of hypercalcemia and **hypercalciuria** is avoided as there is no rapid rise in serum Ca levels.

ACCESSION NUMBER: 1980-82501C [46] WPIDS
TITLE: Topical compsn. for disorders due to decreased calcium transport - contg. vitamin-D2 or -D3 active cpds. or provitamin(s).
DERWENT CLASS: B01 B05
INVENTOR(S): HOLICK, M F; USKOKOVIC, M R
PATENT ASSIGNEE(S): (HOFF) HOFFMANN LA ROCHE INC
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 4230701	A	19801028	(198046)*		

PRIORITY APPLN. INFO: US 1979-22393 19790321; US 1980-203291 19801103

=> file japio

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	27.48	132.93

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-5.01

FILE 'JAPIO' ENTERED AT 13:08:04 ON 03 NOV 2000
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FILE LAST UPDATED: 17 OCT 2000 <20001017/UP>
FILE COVERS 1976 THROUGH JUNE 30, 2000

=> s l8

L26 9 HYPERCALCIURIA

```
=> s l26 and (risk or risk assessment or screening or detection?)
          4433 RISK
          4433 RISK
           196 ASSESSMENT
             2 RISK ASSESSMENT
                 (RISK(W)ASSESSMENT)
          5959 SCREENING
        274593 DETECTION?
L27          0 L26 AND (RISK OR RISK ASSESSMENT OR SCREENING OR DETECTION?)

=> d l26 ti abs ibib tot


L26  ANSWER 1 OF 9  JAPIO  COPYRIGHT 2000 JPO
TI   PREPARATION FOR PREVENTING AND TREATING ABSORPTION BONE DISEASE
AN   1996-301783    JAPIO
AB   PURPOSE: To obtain a safe preparation containing thrombomodulin as an
     active ingredient, acting on bone cells, effective for the absorption
bone
     diseases such as hypercalciuria caused by osteoporosis, myeloma
     and lymphoma, and little in side effects.
     CONSTITUTION: This preparation for preventing and treating the absorption
     bone diseases contains thrombomodulin (hereinafter referred to TM) as an
     active ingredient. The TM is preferably a soluble TM originated from a
     natural human urine or a human soluble TM synthesized by a genetic
     recombination method. The objective preparation is preferably
administered
     at a daily does of 0.1-100.mu.mg/kg as the amount of the TM protein. The
     preparation can continuously be administered by retaining the preparation
     in a living body in the form of an imbedding medicine or an osmotic
     pressure pump in which the preparation is sealed. The human
     urine-originated soluble TM is obtained e.g. by filtering a raw urine
with
     acrylic fibers to adsorb off urokinase in the urine on the fibers,
     desalting and concentrating the passed urine with an ultrafiltration
     membrane, and subsequently successively purifying the product with a DEAE
     cellulose column, a DIP-thrombine-agaro-chromatography, etc.
ACCESSION NUMBER:        1996-301783    JAPIO
TITLE:                   PREPARATION FOR PREVENTING AND TREATING ABSORPTION
                         BONE DISEASE
INVENTOR:                TAKAHASHI YASUO; NAKAYAMA KAZUO
PATENT ASSIGNEE(S):      MOCHIDA PHARMACEUT CO LTD,  JP   (CO  401680)
PATENT INFORMATION:

     PATENT NO    KIND   DATE        ERA     MAIN IPC
     ------------------------------------------------------
     JP 08301783  A      19961119    Heisei  (6) A61K038-00

     JP
APPLICATION INFORMATION
     ST19N FORMAT:       JP1995-113232       19950511
     ORIGINAL:           JP07113232          Heisei
SOURCE:                  PATENT ABSTRACTS OF JAPAN (CD-ROM), Unexamined
                         Applications, Vol. 96, No. 11

L26  ANSWER 2 OF 9  JAPIO  COPYRIGHT 2000 JPO
TI   NEW PHOSPHORIC ACID DERIVATIVE, ITS PRODUCTION AND MEDICINAL COMPOSITION
     CONTAINING THE SAME
AN   1996-225586    JAPIO
AB   PURPOSE: To provide a specific new phosphoric acid derivative containing
a
```

benzazepine ring, etc., having an inhibiting action against neutral endopeptidase and angioensin transferase activit, and useful for preventing and t[redacted]cting hypertension, cardiac fai[redacted]e, angina pectoris, etc.

CONSTITUTION: The new phosphoric acid derivative is represented by formula

I (R is a lower alkyl, an ar(lower)alkyl, hydroxyl group; A is a single bond, an lower alkylene, an alkylenecarbonyl, an amino acid residue, a methyleneamino acid residue, etc.; m is 0, 1; p is 0, 1; q is 1, 2; p+q=2), has an inhibiting activity against neutral endopeptidases and angiotensin transferases, and is useful as an agent for preventing and treating hyper tension, cardiac failure, renal failure, hyperandrostronemia, **hypercalciuria**, hyperleninemia, etc. The compound is obtained by deesterifying an ester derivative of formula II (R1 is a lower alkyl, an ar(lower)alkyl; R2a, R2b are each H, an lower alkyl, an ar(lower)alkyl) by a catalytical hydrogenation reaction in the presence of a Pd catalyst.

ACCESSION NUMBER: 1996-225586 JAPIO
TITLE: NEW PHOSPHORIC ACID DERIVATIVE, ITS PRODUCTION AND MEDICINAL COMPOSITION CONTAINING THE SAME
INVENTOR: TAKIMOTO KOICHI; TAKENAKA KOHEI; OKITSU OSAMU; KOBAYASHI YUIKO
PATENT ASSIGNEE(S): FUJISAWA PHARMACEUT CO LTD, JP (CO 000524)
PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC
JP 08225586	A	19960903	Heisei	(6) C07F009-553

JP
APPLICATION INFORMATION
ST19N FORMAT: JP1995-59765 19950222
ORIGINAL: JP07059765 Heisei
SOURCE: PATENT ABSTRACTS OF JAPAN (CD-ROM), Unexamined Applications, Vol. 96, No. 9

L26 ANSWER 3 OF 9 JAPIO COPYRIGHT 2000 JPO
TI NEW MERCAPTO-AMIDO DERIVATIVE
AN 1996-041015 JAPIO
AB PURPOSE: To obtain a new mercapto-amido derivative and a salt thereof having double inhibitory activity against neutral endopeptidase EC 3.4.24.11 and angiotensinase, thus useful as a medicine.

CONSTITUTION: This mercapto-amido derivative (and a salt thereof) is expressed by formula I (R1 is a lower alkyl, cyclo(lower)alkyl, aryl or heterocycle; R2 is a heterocycle, lower alkyl or H; X is S, -, or lower alkylene; Y is -, or lower alkylene; (n) is 0 or 1), e.g. N-((S)-2-mercaptomethylpentanoyl)-3-(1,3- benzothiazol-2-yl)-(S)-alanine. The compound of formula I is obtained by reaction between a compound of formula II and a compound of formula III. Of the compounds of formula I, a compound of formula IV is esp. preferable. This new compound is useful for preventing and treating various cardiovascular disorders such as hypertension, heart failure and stenocardia, renal failure, periodic edema, hyperaldosteronism, **hypercalciuria**, etc.

ACCESSION NUMBER: 1996-041015 JAPIO
TITLE: NEW MERCAPTO-AMIDO DERIVATIVE
INVENTOR: SHIOKAWA YOICHI; TAKIMOTO KOICHI; TAKENAKA KOHEI; KOBAYASHI YUIKO; OKITSU OSAMU
PATENT ASSIGNEE(S): FUJISAWA PHARMACEUT CO LTD, JP (CO 000524)
PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC

JP 08041015	A	19960213	Heisei	(6) C07C323-52

JP

APPLICATION INFORMATION

ST19N FORMAT:	JP1994-194775	19940727
ORIGINAL:	JP06194775	Heisei

SOURCE: PATENT ABSTRACTS OF JAPAN (CD-ROM), Unexamined Applications, Vol. 96, No. 2

L26 ANSWER 4 OF 9 JAPIO COPYRIGHT 2000 JPO

TI PROPIONAMIDE DERIVATIVE

AN 1993-262709 JAPIO

AB PURPOSE: To obtain a new propionamide derivative useful as a therapeutic agent and/or preventive for cardiovascular disorder, renal insufficiency, periodic edema, hyperaldosteronemia, **hypercalciuria**, etc., having inhibitory action on neutral endopeptidase.

CONSTITUTION: A compound of formula I (R1 and R3 are ar(lower)alkyl; R2 is carboxy, hydroxyaminocarbonyl or (protected hydroxy)aminocarbonyl; R4 is carboxy or protected carboxy; A is lower alkylene or cyclo (lower) alkylene) and its salt such as 3-((2R,3R)-2,3-dibenzyl-3-carboxypropionamido)propionic acid ethyl ester. A compound of Ia (R4a is protected carboxyl) is obtained by reacting a compound of formula II with a compound of formula III or its salt. The compound has inhibitory action on neutral endopeptidase EC3. 4. 24. 11 and is useful for treating and preventing glaucoma, asthma, inflammation, ache, epilepsy, etc.

ACCESSION NUMBER: 1993-262709 JAPIO

TITLE: PROPIONAMIDE DERIVATIVE

INVENTOR: SHIOKAWA YOICHI; TAKIMOTO KOICHI; TAKENAKA KOHEI; MITSUNAGA TAKAFUMI

PATENT ASSIGNEE(S): FUJISAWA PHARMACEUT CO LTD, JP (CO 000524)

PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC
JP 05262709	A	19931012	Heisei	(5) C07C233-51

JP

APPLICATION INFORMATION

ST19N FORMAT:	JP1992-159162	19920618
ORIGINAL:	JP04159162	Heisei

PRIORITY APPLN. INFO.: GB1991 9114006 19910628

SOURCE: PATENT ABSTRACTS OF JAPAN, Unexamined Applications, Section: C, Sect. No. 1154, Vol. 18, No. 32, P. 112 (19940118)

L26 ANSWER 5 OF 9 JAPIO COPYRIGHT 2000 JPO

TI VITAMIN D3 METABOLITE PREPARATION

AN 1991-058934 JAPIO

AB PURPOSE: To provide a preparation effective for Ca dysbolisms such as hypercalcemia, **hypercalciuria** and osteoporosis, comprising 24,25- dihydroxycholecalciferol and sesame oil.

CONSTITUTION: 0.00001-1.0wt.% (preferably 0.001-0.1) of 24,25-dihydroxycholecalciferol (24,25-(OH)2-D3) is dissolved in sesame oil to provide the subject preparation. An oil squeezed from sesame all can be used as the sesame oil but an oil having a peroxide value of 0-2.1me/kg (preferably 0-1.0me/kg) is especially preferable. The 24,25-(OH)2-D3 includes 24R,25- and 24S, 25- isomers and may be dissolved in the sesame oil after dissolved in an organic solvent. The preparation may be compounded if necessary with a flavor, aromatic agent, thickener, preservative, etc.

ACCESSION NUMBER: 1991-058934 JAPIO

TITLE: VITAMIN D3 METABOLITE PREPARATION
INVENTOR: HIRAI TORU; NIIMURA KOICHI; YAMATO HIDEYUKI
PATENT ASSIGNEE(S): KUREHA CHEM IND CO LTD, JP (CO 000110)
PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC
JP 03058934	A	19910314	Heisei	(5) A61K031-59

JP
APPLICATION INFORMATION
ST19N FORMAT: JP1989-195662 19890728
ORIGINAL: JP01195662 Heisei
SOURCE: PATENT ABSTRACTS OF JAPAN, Unexamined Applications, Section: C, Sect. No. 835, Vol. 15, No. 2, P. 89 (19910527)

L26 ANSWER 6 OF 9 JAPIO COPYRIGHT 2000 JPO
TI PEPTIDE RELATING TO PTHRP, PREPARATION AND USE THEREOF
AN 1990-207099 JAPIO
AB NEW MATERIAL: A polypeptide represented by formula, etc., not having any human parathyroid hormone-relating protein(PTHrP) activity and having an antagonistic activity against the physiological action of the human PTHrP or a peptide having the human PTHrP activity.
USE: A calcium metabolish remedy or a **hypercalciuria** remedy.
PREPARATION: For example, a PTHrP gene is divided with a restriction enzyme into PTHrP(A) and PTHrP(B), which are synthesized into DNA fragments by a phosphoamidite method, respectively, and subsequently converted into genes corresponding to partial peptides by a ligation reaction. The genes are inserted into cloning vectors, and Escherichia coli transformed with the vectors is cultured. Plasmids are extracted from
the train to give pCU-PTHrP(A) and (B), which are treated with a restriction enzyme and combined with expression vectors. Escherichia coli is transformed with the treated expression vectors and the transformed strain is cultured, followed by providing a polypeptide from the cultured product.
ACCESSION NUMBER: 1990-207099 JAPIO
TITLE: PEPTIDE RELATING TO PTHRP, PREPARATION AND USE THEREOF
INVENTOR: URAGAMI KENICHI; MIKI KEIZABURO
PATENT ASSIGNEE(S): TONEN CORP, JP (CO 352374)
PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC
JP 02207099	A	19900816	Heisei	(5) C07K007-10

JP
APPLICATION INFORMATION
ST19N FORMAT: JP1989-28023 19890207
ORIGINAL: JP01028023 Heisei
SOURCE: PATENT ABSTRACTS OF JAPAN, Unexamined Applications, Section: C, Sect. No. 774, Vol. 14, No. 497, P. 118 (19901030)

L26 ANSWER 7 OF 9 JAPIO COPYRIGHT 2000 JPO
TI REMEDY AND PREVENTIVE FOR **HYPERCALCIURIA**
AN 1987-029526 JAPIO
AB PURPOSE: To obtain the titled agent by neutralizing an acidic aqueous solution of a phytic acid salt in the presence of a component selected from amino acids, peptides and water-soluble proteins, and using the resultant precipitate as an active component.

CONSTITUTION: An acidic aqueous solution of a phytic acid salt (e.g. a double salt of phytic acid and a cation composed mainly of magnesium and potassium) is neutralized in the presence of one or more components selected from amino acids (e.g. glycine, alanine, valine, methonine, serine, asparagin, etc.), peptides (e.g. glycylglycine, glycylglycylglycine, etc.) and water-soluble proteins (e.g. albumin, casein, etc.), and the precipitate is used as an active component of the objective remedy and preventive for **hypercalciuria**. It is administered in the form of powder, tablet, etc., at a dose of 600-800mg in terms of the phytic acid salt orally after meal.

ACCESSION NUMBER: 1987-029526 JAPIO
TITLE: REMEDY AND PREVENTIVE FOR **HYPERCALCIURIA**
INVENTOR: AKIBA KIYOSHI; IZUMIYA SHOICHI; KOBAYASHI TOKIO
PATENT ASSIGNEE(S): WAKAMOTO PHARMACEUT CO LTD, JP (CO 351728)
PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC
JP 62029526	A	19870207	Showa	(4) A61K031-66

JP
APPLICATION INFORMATION
ST19N FORMAT: JP1985-168610 19850801
ORIGINAL: JP60168610 Showa
SOURCE: PATENT ABSTRACTS OF JAPAN, Unexamined Applications, Section: C, Sect. No. 433, Vol. 11, No. 2, P. 45 (19870703)

L26 ANSWER 8 OF 9 JAPIO COPYRIGHT 2000 JPO
TI CALCIUM ADJUSTING AGENT CONTAINING 25-HYDROXYVITAMIN D3-26,23-PEROXYLACTONE AS ACTIVE CONSTITUENT
AN 1984-065014 JAPIO
AB NEW MATERIAL: 25-Hydroxyvitamin D3-26,23-peroxylactone expressed by the formula.
USE: An orally administrable calcium adjusting agent, capable of reducing the calcium level in the blood serum, and useful for treating hypercalcemia caused by the administration of vitamin D analogs, hypercalcemia caused by malignant tumors and **hypercalciuria**, hyperthyroidism and Behcet's disease, etc.
PREPARATION: min D3 or 25-hydroxyvitamin D3 in an amount within the toxically acceptable range is administered to an animal, e.g. domestic fowl or pig, etc. by a means, e.g. oral, intravenous or intramuscular injection, etc. About 2-50hr after the administration, the compound expressed by the formula as a metabolite of the vitamin D3 or 25-hydroxyvitamin D23 is isolated and extracted from a fat-soluble portion in the blood serum of the above-mentioned animal by the column chromatography, etc.

ACCESSION NUMBER: 1984-065014 JAPIO
TITLE: CALCIUM ADJUSTING AGENT CONTAINING 25-HYDROXYVITAMIN D3-26,23-PEROXYLACTONE AS ACTIVE CONSTITUENT
INVENTOR: ISHIZUKA SEIICHI; KUBO YORITSUGU
PATENT ASSIGNEE(S): TEIJIN LTD, JP (CO 000300)
PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC
JP 59065014	A	19840413	Showa	(3) A61K031-365

JP
APPLICATION INFORMATION
ST19N FORMAT: JP1982-175265 19821007
ORIGINAL: JP57175265 Showa

SOURCE: PATENT ABSTRACTS OF JAPAN, Unexamined Applications, Section: C, Sect. No. 235, Vol. 8, No. 1621, P. 151 (19840726)

L26 ANSWER 9 OF 9 JAPIO COPYRIGHT 2000 JPO
TI CALCIUM ADJUSTING AGENT CONTAINING 25-HYDROXYVITAMIN D3-26, 23-LACTONE
AN 1983-118515 JAPIO
AB PURPOSE: A calcium adjusting agent, containing 25-hydroxyvitamin D3-26,23-
lactone as an active constituent, having the activity of lowering the calcium concentration in the blood serum, and useful for treating hypercalcemia, **hypercalciuria**, etc.
CONSTITUTION: A calcium adjusting agent containing 25-hydroxyvitamin D3-26, 23-lactone as an active constituent. The compound is a compound expressed by the formula, and 23(S)25(R)-25-hydroxyvitamin D3-26,23-lactone which is a natural substance is suitable due to the preferred physiological activity thereof. The compound is capable of reducing the calcium content in the blood serum, and useful for treating diseases, e.g. hypercalcemia, hypercalcemia due to malignant, tumor, **hypercalciuria**, hyperfunction of accessory thyroid, Behccet's disease, etc., caused by 1.alpha.,25-dihydroxycholecalciferol, etc. administered in treating osteoporosis, osteomalacia, etc.
ACCESSION NUMBER: 1983-118515 JAPIO
TITLE: CALCIUM ADJUSTING AGENT CONTAINING 25-HYDROXYVITAMIN D3-26, 23-LACTONE
INVENTOR: ISHIZUKA SEIICHI; KUBO YORITSUGU
PATENT ASSIGNEE(S): TEIJIN LTD, JP (CO 000300)
PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC
JP 58118515	A	19830714	Showa	(3) A61K031-59

JP
APPLICATION INFORMATION
ST19N FORMAT: JP1981-213108 19811229
ORIGINAL: JP56213108 Showa
SOURCE: PATENT ABSTRACTS OF JAPAN, Unexamined Applications, Section: C, Sect. No. 188, Vol. 7, No. 2191, P. 163 (19830929)

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=> s l8

L28 125 HYPERCALCIURIA

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=> s l28 and (risk or risk assessment or detection or screening?)
         35301 RISK
         35301 RISK
         28731 ASSESSMENT
          1001 RISK ASSESSMENT
                 (RISK(W)ASSESSMENT)
         78407 DETECTION
         26217 SCREENING?
L29         17 L28 AND (RISK OR RISK ASSESSMENT OR DETECTION OR SCREENING?)

=> d l29 ti abs ibib tot


L29  ANSWER 1 OF 17  JICST-EPlus COPYRIGHT 2000 JST
TI   A Case With Hypercalcemic Nephropathy Due to Long-Term Vitamin D And
     Calcium Administration after Total Parathyroidectomy.
AB   A patient with renal injury administrated high-dose active vitamin D and
     calcium for long period of time after total thyro-parathyroidectomy due
to
     thyroid cancer is reported. The patient was a 51-year-old woman treated
by
     thyroxin, alfacalcidol 3.0.MU.g/day and calcium after surgery. Her renal
     function deteriorated four years later, to a serum creatinine level of
1.9
     mg/dl and creatinine clearance to 38.7 ml/min. Tubular injury was
     especially severe, with a Fishberg's test value of 1013 mosm/kg, urine
     .BETA.2microglobulin of 1369 .MU.g/l and urine NAG/Cr index of 7.24
     U/gvCr. Urine Ca excretion increased to 183 mg/day (hypercalciuria
     ). CT reverled a rigt renal calcium stone. The glomeruli were intact
     histologically and tubulointerstitial damage was mainly observed with
     minimal calcification in tubular cells, so-called hypercalcemic
     nephropathy. She was given alfacalcidol 0.5 .MU.g /day orally, which
     maintained low serum calcium levels. Serum creatinine level subsequently
     decreased to 0.9 mg/dl (normal level). For maintenance of serum calcium
     level in non-PTH patients, it is sufficient to administer low-dose
vitamin
     D. Combination therapy with vitamin D and long-term calcium
administration
     may present the risk of hypercalcemia and renal dysfunction.
     (author abst.)
ACCESSION NUMBER:   1000263140  JICST-EPlus
TITLE:              A Case With Hypercalcemic Nephropathy Due to Long-Term
                    Vitamin D And Calcium Administration after Total
                    Parathyroidectomy.
AUTHOR:             UEDA TAKAHIRO; SAKURAI TETSUO; SHIROSHITA KOICHI; FUKAZAWA
                    SACHIKO; OKAMOTO NOBUHIKO; UEBAYASHI MINORU; SATO
HIDETOSHI
                    KONNO NORIMICHI
CORPORATE SOURCE:   Sapporo City Hosp.
                    Konnonaikakurinikku
SOURCE:             Shiritsu Sapporo Byoin Ishi (Acta Medica Nosocomi
Sapporo),
                    (1999) vol. 59, no. 2, pp. 155-159. Journal Code: F0663A
                    (Fig. 8, Tbl. 1, Ref. 8)
                    CODEN: SSBIAF; ISSN: 0288-6073
PUB. COUNTRY:       Japan
DOCUMENT TYPE:      Journal; Short Communication
LANGUAGE:           Japanese
STATUS:             New

L29  ANSWER 2 OF 17  JICST-EPlus COPYRIGHT 2000 JST
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TI Antioxidant Status and Urinary Stone **Risk** Parameters in Essential Hypertension.

ACCESSION NUMBER: 1[redacted]159356 JICST-EPlus
TITLE: Antioxidant Status and Urinary Stone **Risk** Parameters in Essential Hypertension.
AUTHOR: LATHA E; HARIPRASAD C; ANBAZHAGAN M
SUNDARI R; NANDINI S
SELVAM R
CORPORATE SOURCE: Srmc And Ri(du), Chennai, Ind
Madras Univ., Chennai, Ind
E.s.i. Hospital, Chennai, Ind
SOURCE: J Clin Biochem Nutr, (1999) vol. 27, no. 1, pp. 49-54.
Journal Code: X0851A (Tbl. 4, Ref. 37)
ISSN: 0912-0009
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: English
STATUS: New

L29 ANSWER 3 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Mineral metabolism and bone tissue in rats with streptozotocin-induced diabetes.
AB We Investigated mineral homeostasis and bone morphologic changes in rats with diabetes mellitus induced by 8-week treatment with streptozotocin (STZ-diabetic rats) versus normal rats. STZ-diabetic rats showed necrosis of pancreatic .BETA.-cells, persistent hyper-glycemia, and depression of weight gain. In chronic phase STZ-diabetic rats, there were moderate hypercalcemia and hyperphosphatemia, with marked **hypercalciuria** and hyperphosphaturia. These rats were hypomagnesemic and showed high urinary magnesium concentration. Serum zinc level remained unaltered while urinary zinc excretion was increased. Histrogic examination of the ilium, vertebra, femur and tibia revealed anextremely scant stromal ost, diminished compact bone and trabeculae, and enlarged marrow cavity in the STZ-diabetic rats. The above findings indicate that the metabolic abnormalities in STZ-diabetic rats may constitute a **risk** factor for osteoporosis. (author abst.)

ACCESSION NUMBER: 980264194 JICST-EPlus
TITLE: Mineral metabolism and bone tissue in rats with streptozotocin-induced diabetes.
AUTHOR: TERAKI YOSHIMI
UCHIUMI AKIRA
CORPORATE SOURCE: Yakushimatokushukaibyoin
National Inst. Materials and Chemical Res.
SOURCE: Biomed Res Trace Elem, (1997) vol. 8, no. 3, pp. 209-210.
Journal Code: L1046A (Fig. 2, Ref. 2)
ISSN: 0916-717X
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Short Communication
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 4 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Clinical significance of hypocitraturia in kidney stone patients.
AB Although a low concentration of urinary citrate is cited as one of the **risk** factors promoting stone formation or recurrence among patients with urinary stones, its clinical significance remains obscure. We studied 62 kidney stone patients with a low urinary citrate excretion (hypocitraturia) of less than 320mg/day, without any apparent cause. The incidence of hypocitraturia in 722 kidney stone patients followed up at our stone clinic was 14.6%. Among the 62 patients, 37 had an uncomplicated hypocitraturia as the sole abnormality, while 25 had other associated

stone **risk** factors, including **hypercalciuria** in 11% (7/62), hyperuricosuria in 24% (15/ 62), hyperoxaluria in 5% (3/62) and hypomagnesuria in 24% (15/62). The rate of urinary stone recurrence was 38% (14/37) in uncomplicated hypocitraturia, and 52% (13/25) in complicated hypocitraturia, but no statistical difference was observed. Regarding the outcome of stones, more stones were managed with lithotripsy and more passed spontaneously in the hypocitraturic patients than in the control patients with normal urinary citrate excretion. The diagnosis of hypocitraturia complicated with additional stone risks urged us to treat patients more vigorously with lithotripsy and medication, resulting in a prompt cure. (author abst.)

ACCESSION NUMBER: 940057773 JICST-EPlus
TITLE: Clinical significance of hypocitraturia in kidney stone patients.
AUTHOR: MATSUSHITA K; TANIKAWA K; MASUDA A MATSUZAKI S
CORPORATE SOURCE: Tokai Univercity Tokyo Hospital, Tokyo, JPN
Inagi Municipal Hospital, Tokyo, JPN
SOURCE: Nihon Jinzo Gakkaishi (Japanese Journal of Nephrology), (1993) vol. 35, no. 11, pp. 1253-1257. Journal Code: Z0142A
(Tbl. 5, Ref. 7)
ISSN: 0385-2385
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: English
STATUS: New

L29 ANSWER 5 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Two cases of idiopathic **hypercalciuria**.
AB Idiopathic **hypercalciuria**(IH) is defined as an abnormally high urine calcium excretion rate without original diseases that cause **hypercalciuria** such as hyperparathyroidism, renal tubular acidosis, vitamin D intoxication, or Cushing syndrome. IH is well-known as one of the causes of urolithiasis in adults. IH has been made much account of as the cause of hematuria in childhood, since Stapleton and his colleague reported **hypercalciuria** in children with hematuria in 1984. Two girls who had asymptomatic hematuria were diagnosed as IH by the measurement of 24-hour urinary calcium excretion after the **screening** of measuring the calcium/creatinine concentration ratio in early morning urine. Both cases were judged as absorptive idiopathic **hypercalciuria** by means of calcium-loading test. They were treated by only mild restriction of salt or calcium and have had no gross hematuria episode after treatment. (author abst.)

ACCESSION NUMBER: 930689531 JICST-EPlus
TITLE: Two cases of idiopathic **hypercalciuria**.
AUTHOR: SHIMA MASAYUKI; MORIMOTO HIROYUKI; MINOWA HIDEKI; YAMASHITA RYUJI; KAMITSUJI HIDEKAZU
CORPORATE SOURCE: Nara Prefect. Nara Hospital
SOURCE: J Nara Med Assoc, (1993) vol. 44, no. 2, pp. 130-135. Journal Code: F0524A (Fig. 4, Tbl. 2, Ref. 15)
CODEN: NAIZAM; ISSN: 0469-5550
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 6 OF 17 JICST-EPlus COPYRIGHT 2000 JST

TI Metabolic Evaluation of Stone Patients Treated by ESWL.
AB A recurrence rate of 40 to 50% in urinary stone disease warrants a metabolic evaluation of stone patients for stone-forming **risk** factors, even in the extracorporeal shock wave lithotripsy (ESWL) era. A **screening** test, including a blood biochemistry test and 24-hour urine analysis, for the **detection** of metabolic abnormalities was conducted on 252 stone patients, one day before ESWL treatment, in an inpatient setting. Hypercalcemia, hyperuricemia, **hypercalciuria** and hyperuricosuria were found in 9(3.6%), 40(15.9%), 42(16.7%) and 18(7.1%) patients, respectively. A total of 90 patients (35.7%) had at least one of these abnormalities. Primary hyperparathyroidism was subsequently disclosed in 2 patients by further examinations. These results suggest that a **screening** test in this manner is practical and applicable, even in the ESWL era. A satisfactory metabolic evaluation can be achieved in one day which indicates those patients who need further evaluation. (author abst.)

ACCESSION NUMBER: 930598015 JICST-EPlus
TITLE: Metabolic Evaluation of Stone Patients Treated by ESWL.
AUTHOR: FUJIMOTO NOBUMASA; KYO MASAHIRO; ICHIKAWA YASUJI; NAGANO SHUNSUKE
CORPORATE SOURCE: Hyogo Prefect. Nishinomiya Hospital
SOURCE: Nishinippon Hinyokika (Nishinihon Journal of Urology), (1993) vol. 55, no. 6, pp. 833-836. Journal Code: Z0253B (Tbl. 4, Ref. 7)
ISSN: 0029-0726
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 7 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI UItrasonic findings in urogenital system and urinary Ca/Cr ratio in children with hematuria.
AB To evaluate the usefulness of ultrasonogoaphic examination in urogenital system and the measurement of urinary concetration of Calcium /Creatinine(Ca/Cr) ratio for the **screening** of urgenital pathology, the incidence of positice ultrasonographic findings in hematuric childern vs. nonhematuric(control) children (N=361 vs. N=532) and urinary Ca/Cr ratio in 3 year-old children (N=1023) with or without hematuria detected by dipstick paper test at the health check for 3 year-old children were studied. 1)Ultrasonography showed the findings indicating urolithiasis in 24 of 361 (6.6%) hematuric children and 4 of 532 (0.7%) nonhematuric (control) children (highly significant difference between two groups). Other abnormal findings (pelvic or ureteral dilataion etc.) were detercted in 19 of 361(5.3%) in hematuric children and 17 of 532(3.2%) nonhematuric (control) children (no significant difference). 2) Urinary Ca/Cr ratios in 3 year-old hematuric and nonhematuric (conotrol) children reverlaed no significant difference between those two groups(0.10.+-.0.08 vs. 0.11.+-.0.10). 3) Incience of **hypercalciuria**(urinary Ca/Cr ratios higher than 0.21) in hematuric and nonhematuric children were 4 of 71(5.6%) and 113 of 952(11.9%) (no significant difference). (author abst.)

ACCESSION NUMBER: 920101439 JICST-EPlus
TITLE: UItrasonic findings in urogenital system and urinary Ca/Cr ratio in children with hematuria.
AUTHOR: ISHIKAWA YUTAKA
CORPORATE SOURCE: Kurume Univ., Faculty of Medicine
SOURCE: Kurume Igakkai Zasshi (Journal of the Kurume Medical Association), (1991) vol. 54, no. 9, pp. 562-571. Journal Code: F0979B (Fig. 8, Tbl. 6, Ref. 53)
CODEN: KIZAAL; ISSN: 0368-5810
PUB. COUNTRY: Japan

DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 8 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Bone mineral densitometry by dual photon absorptionetry in patients with urolithiasis. On the possibility of the differential diagnosis of idiopathic **hypercalciuria**.
AB Bobe mineral density(BMD) of the 3rd lumbar spine was measured by dual photon absorptiometry(DPA) in 8 patients with primary hyperparathyroidism(PHP) and 39 patients with idiopathic urolithiasis(IU).
Of the patients, 15 were classified into idiopathic **hypercalciuria** (IH) which were further classified into 2 type of IH-renal **hypercalciuria**(RH) and absorptive **hypercalciuria**(AH)-by Ca restriction and load test. BMD of the IH patients tended to be lower than patients with normocalciuria, but significantly higher than the PHP patients. BMD of the RH patients was significantly lower than the AH patients. In concluction, DPA may be a simple method for classifying the types of idiopathic **hypercalciuria**. (author abst.)
ACCESSION NUMBER: 920082177 JICST-EPlus
TITLE: Bone mineral densitometry by dual photon absorptionetry in patients with urolithiasis. On the possibility of the differential diagnosis of idiopathic **hypercalciuria**.
AUTHOR: TAKEDA MASAYUKI; KATAYAMA YASUSHI; GO HIDETO; WAKATSUKI SHUNJI; TSUTSUI TOSHIKI; KAWASAKI TAKASHI; SATO SHOTARO; ODANO IKUO
CORPORATE SOURCE: Niigata Univ., School of Medicine
SOURCE: Nippon Hinyokika Gakkai Zasshi (Japanese Journal of Urology), (1991) vol. 82, no. 12, pp. 1954-1958. Journal Code: Z0766A (Fig. 7, Tbl. 2, Ref. 11)
ISSN: 0021-5287
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 9 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Preparation of a standard diet for out-patients for studies of lithogenesis.
AB With the development of extracorporeal shock wave lithotripsy treatment, the duration of hospitalization for stone patients fortunately has become shorter. However, a detailed analysis of lithogenesis is not possible during such patients' short hospital stays. We prepared a standard diet to be eaten at home for investigation of liGhogenesis at the out-patient clinic. This diet was nutritionally well-balanced and contained the following: energy:2000Kcal, total protein:70-75g, animal protein:30-35g, carbohydrate:510g, fat and oil:50-60g, calcium:600-630mg and magnesium:320mg. The urine of 24 male patients with stones on a free diet and the same patients after 3 days on the standard diet was analyzed for urea-nitrogen, uric acid, sodium, calcium, phosphorus, magnesium, citric acid and oxalic acid. The reaults were compared with those in 17 healthy male subjects who were eating the standard diet (controls). It was found that 66% of **hypercalciuria** (>=300mg/day) on a free diet became normocalciuria on the standard diet. The **hypercalciuria** was therefore thought to be of dietary origin. Moreover, urinary excretion of urea nitrogen, uric acid, sodium and phosphorus by patients remarkably decreased after 3 days on the standard diet, which was not different from that of controls. These results suggest that the standard diet at home is useful in the **screening** of **hypercalciuria** and also quite adequate for patients with stones. (author abst.)

ACCESSION NUMBER: 910339285 JICST-EPlus
TITLE: Preparation of a standard diet for out-patients for studies of lithogenesis.
AUTHOR: IGUCHI MASANORI; UMEKAWA TOORU; KIWAMOTO HIRO KATAYAMA YOSHIKAZU; ISHIKAWA YASUAKI; KODAMA MITSUMASA; TAKAMURA CHISATO; TAKADA MASAHIKO; KURITA TAKASHI
CORPORATE SOURCE: Kaizuka Municipal City Hospital
Kinki Univ., Faculty of Medicine
SOURCE: Nippon Hinyokika Gakkai Zasshi (Japanese Journal of Urology), (1991) vol. 82, no. 3, pp. 378-387. Journal Code: Z0766A (Fig. 10, Tbl. 4, Ref. 10)
ISSN: 0021-5287
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 10 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI **Screening** for **hypercalciuria**.
AB Reference values for the urinary calcium/creatinine ratio(Ca/Cr ratio) in the first morning urine were established in 361 healthy children aged 5 to 15 years, on unrestricted diets. The urinary Ca/Cr ratio in the urine upon arising was independent of sex but dependent upon age. The measurement of the urinary Ca/Cr ratio in the urine upon arising while on unrestricted diets may be a reasonable **screening** test for elevated calcium excretion. On the basis of the urinary Ca/Cr ratios in the urine upon arising during unrestricted diets and the calciuric response to calcium restricted diets and the oral calcium loading test, idiopathic **hypercalciuria**(IH) was subclassified into three groups: (1) absorptive **hypercalciuria**; (2) renal **hypercalciuria**; (3) dietary **hypercalciuria**. The pathogenesis of IH is controversial. Our data suggest that disordered 1,25(OH)2 vitamin D metabolism with excessive urinary phosphate excretion occurs in absorptive **hypercalciuria**. (author abst.)
ACCESSION NUMBER: 910151869 JICST-EPlus
TITLE: **Screening for hypercalciuria**.
AUTHOR: AKASHI S; MOTIZUKI H
CORPORATE SOURCE: Saitama Children's Medical Center, Saitama
SOURCE: Acta Paediatr Jpn, (1990) vol. 32, no. 6, pp. 701-709. Journal Code: Z0373B (Fig. 6, Tbl. 2, Ref. 24)
ISSN: 0374-5600
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: English
STATUS: New

L29 ANSWER 11 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Reference value for urinary calcium excretion in normal children for **screening** of **hypercalciuria**.
AB We collected early morning fasting and second void (F) and postprandial(Po) urine samples in 1,192 school children and measured urinary calcium(Ca), phosphrous(P), sodium(Na) and creatinine(Cr) to assess the normal value of urinary calcium excretion adn the relationship between Ca excretion and Na, P excretions in creatinine ratio. The results were as follows; 1) The FCA/Cr and PoCa/Cr distributions were both logarithmic forms. They had good correlation (r=0.755), and their mean values decreased by age from 11 year to 14 years old. The distribution

pattern of Ca excretion changed to have a peak when 11 years old in the unit of mg/g IGF-2) FCa/Cr had good correlation with FNa/Cr (r=0.514, p<0.01), and weak correlation with FP/Cr (r=0.287, p<0.05). The hypercalciuric group (FCa/Cr.GEQ.0.21) had significantily higher FNA/Cr value than the normocalciuric group (FCa/Cr<0.21). 3) We had only one case
that had both hypercaliciuria (FCa/Cr.GEQ.0.21 or PoCa/Cr.GEQ.0.28) and hematuria. But the hypercaliciuric group had many more cases of renal stone formation in their family members than normocalciuric group (9.6% vs
3.6%). (author abst.)

ACCESSION NUMBER: 900347676 JICST-EPlus
TITLE: Reference value for urinary calcium excretion in normal children for **screening** of **hypercalciuria**
.
AUTHOR: NISHIOKA TADASHI; UDAGAWA JUNKO; KURAYAMA HIDEAKI TAKAYANAGI NAOKO; NAKAMURA FUMIKO; NISHIMUTA TOSHIYUKI; MORI KAZUO
YASUDA TOSHIYUKI; NIIMI HIROO
CORPORATE SOURCE: National Sanatorium Chiba Higashi Hospital
National Sanatorium Shimo-Shizu Hospital
Chiba Univ.
SOURCE: Nippon Shonika Gakkai Zasshi (Journal of the Japan Pediatric Society), (1990) vol. 94, no. 2, pp. 264-269.
Journal Code: F0896A (Fig. 6, Tbl. 3, Ref. 19)
CODEN: NIPOAC; ISSN: 0001-6543
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 12 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Clinical study on 32 patients who underwent parathyroidectomy at Osaka cityuniversity hospital.
AB We retrospectively reviewed 32 patients who underwent parathyroidetomy at our hospital for the last fourteen years. 1) Clinical appearance of primary hyperparathyroidism was in younger age in women. 2) In previous history or at the time of PTX, 9 patients had malignant tumors including 6
thyroid cencers, 36% of the patients with out bone related symptoms had a remarkable decrease in bone mineral content. 3) After PTX, none of patients had recurrent urolithiasis and bone mineral content of all patients was significantly increased in a short time. In addition, upper GI complaints were improved, or hypertension was partially normalized. However, renal insufficiency remained unchanged. 4) In preoperative localization study, Ultrasound sonography (US) demonstrated the best accurracy rate of 88% when only one gland was involved. US was able to detect multiple gland involvement only in 20% of 5 cases. 5) **Hypercalciuria** was recognized as one of the **risk** factors of stone formation in patients with primary hyperparathyroidism. (author abst.)

ACCESSION NUMBER: 900336885 JICST-EPlus
TITLE: Clinical study on 32 patients who underwent parathyroidectomy at Osaka cityuniversity hospital.
AUTHOR: SAKAMOTO WATARU; KISHIMOTO TAKETOSHI; NISHISAKA MASAYASU; IIMORI HIRONORI; MAEKAWA MASANOBU; SUKANO SEIJI; UMEYAMA KAORU; NISHIZAWA YOSHIKI; MORII HIRONORI
CORPORATE SOURCE: Osaka City Univ., Medical School
SOURCE: Nippon Hinyokika Gakkai Zasshi (Japanese Journal of Urology), (1990) vol. 81, no. 2, pp. 230-235. Journal Code:
Z0766A (Fig. 2, Tbl. 3, Ref. 13)
ISSN: 0021-5287

PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 13 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Two cases of idiopathic **hypercalciuria** with asymptomatic hematuria.
ACCESSION NUMBER: 890581975 JICST-EPlus
TITLE: Two cases of idiopathic **hypercalciuria** with asymptomatic hematuria.
AUTHOR: UCHINO TAKAKO; WAKAMATSU RIE; FURUSE AKIO
CORPORATE SOURCE: National Sanatorium Nishi Beppu Hospital
SOURCE: Shonika Shinryo (Journal of Pediatric Practice), (1989) vol. 52, no. 7, pp. 1546-1549. Journal Code: Z0217A (Tbl. 3, Ref. 5)
ISSN: 0386-9806
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 14 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Influence of dietary animal protein on renal stone disease.
AB The effect of dietary protein load on the incidence of nephrolithiasis was studied in rats and men. Three groups of adult male Wister rats were fed with a standard protein diet, a high protein diet, or a low protein diet for 4 weeks. In the high protein group, calcium excretion was significantly increased and citrate excretion was remarkably decreased. This group also exhibited low grade metabolic acidosis due to catabolism of excess amino acids, and increases in urinary cyclic AMP excretion and bone resorption. These findings indicate that protein-induced **hypercalciuria** is due to low grade metabolic acidosis, which directly affects renal handling of calcium. Long-term calcium loss in the urine may lead to negative calcium balance and hyperfunction of the parathyroid gland may induce bone resorption. The influence of 40g animal protein load on urinary **risk** factors of calcium stone formation was investigated in 23 healthy males and 26 patients with nephrolithiasis.
All subjects were given control diets each day containing 60g protein for a week and during the next week each received an additional 40g animal protein. In the controls, added dietary protein resulted in decreased urinary citrate and increased urinary uric acid, with no change in urinary calcium or cyclic AMP excretion. In contrast, the patients showed increased urinary calcium and cyclic AMP as well as decreased urinary citrate. Further examination of the patients revealed that the significant increases of calcium and cyclic AMP excreton occurred only in hypercalciuric patients, who seemed to be classified into renal **hypercalciuria**. These results suggest that even 40g animal protein affects citrate and uric acid metabolism in normal subjects and patients, and affects calcium metabolism in hypercalciuric patients. These findings indicate the importance of diet guidance to patients with nephrolithiasis, with special regard to the correction of excessive animal protein intake.(author abst.)
ACCESSION NUMBER: 890418127 JICST-EPlus
TITLE: Influence of dietary animal protein on renal stone disease.
AUTHOR: KATOH YOSHINARI
CORPORATE SOURCE: Kinki Univ., Faculty of Medicine

SOURCE: Nippon Hinyokika Gakkai Zasshi (Japanese Journal of Urology), (1989) vol. 80, no. 6, pp. 823-831. Journal Code: Z0766A (Fig. 6, Tbl. 3, Ref. 27)
ISSN: 0021-5287
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

L29 ANSWER 15 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Idiopathic **hypercalciuria** in children Part 1. Initial **screening** of idiopathic **hypercalciuria** with hematuria and/or urinary stone.
AB Reference values were assessed for **screening** of idiopathic **hypercalciuria** with hematuria and/or urinary stone(Symptomatic idiopathic **hypercalciuria**, SIH) using Ca/Cr ratio of first morning urine specimens(U0Ca/Cr). Tentatively, the initial **screening** was performed by demonstrating the average U0Ca/Cr of 7 days to be greater than 0.21. A definitive diagnosis was made to have the three conditions of 1) the presence of hematuria and/or urinary stones, 2) urinary Ca/Cr ratio after Ca loading test to be greater than 0.27, and 3) resolving hematuria during anticalciuric therapy. A total of 25 patients were screened, of which 18 were confirmed as SIH. The others were not confirmed and suggested as dietary **hypercalciuria** by our diagnostic oriteria. U0Ca/Cr of the the patients with SIH were ranged from 0.21 to 0.50. On the other hand, the average U0Ca/Cr of 7 days for 181 normal children was 0.15.+-.0.08(mean.+-.1 SD). If **screening** of SIH was performed by demonstrating U0Ca/Cr to be greater than 0.23 or 0.31(which was mean.+-.1 SD or mean.+-.2 SD of those of normal children), 4 or 13 patients with confirmed SIH were excepted. We conclude that our criteria of initial **screening**(U0Ca/Cr to be greater than 0.21) is reasonable in the evaluation of children with SIH.(author abst.)
ACCESSION NUMBER: 890205695 JICST-EPlus
TITLE: Idiopathic **hypercalciuria** in children Part 1. Initial **screening** of idiopathic **hypercalciuria** with hematuria and/or urinary stone.
AUTHOR: MOCHIZUKI HIROSHI; KATAYAMA AKIRA; MURAMATSU YASUO; TAHARA HIROFUMI; AKASHI SHUNJI
USUI NOBUO
CORPORATE SOURCE: Saitama Children's Medical Center
Jikei Univ. School of Medicine
SOURCE: Nippon Shonika Gakkai Zasshi (Journal of the Japan Pediatric Society), (1988) vol. 92, no. 9, pp. 1846-1851. Journal Code: F0896A (Fig. 4, Tbl. 1, Ref. 13)
CODEN: NIPOAC; ISSN: 0001-6543
PUB. COUNTRY: Japan
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TI Clinical studies of citrate therapy on urolithiasis. Estimations of urinary citrate excretion in patients with urolithiasis and the results of treatment with sodium-potassium citrate.
AB 1. Calcium stone formers (male; 170, female; 61) and healthy controls (male; 72, female; 37) were examined with respect to urinary citrate and several urinary biochemistries. Urinary citrate was determined using an enzymatic method which we had reported. The following results were obtained: 1) The mean value of urinary citrate excretion was

383.9.+-.156.5mg/day in male controls, and 452.6.+-.171.4mg/day in female controls. In healthy controls, urinary citrate in females was significantly higher than in males ($p<0.05$). 2) In female patients with calcium urolithiasis urinary citrate was significantly lower than the healthy controls. In female patients urinary citrate excretion was significantly lower only in the group of recurrent stone formers. 3) Hypocitraturia was defined when citrate excretion was under 200mg/day in males and under 250mg/day in female. According to this definition, 45 of 116 male patients (26.5%) and 17 of 61 female patients (27.9%) were classified to hypocitraturia. 4) Hypocitraturia was associated with **hypercalciuria** in 12.4 per cent of male stone formers, and in 6.6 per cent of female stone formers. Both of hypocitraturia and hyperoxaluria were found in only about 10 per cent of stone formers in both sexes. These results showed that hypocitraturia itself was one of the serious **risk** factors of stone formation. 5) There were low statistical correlation between urinary citrate and urine volume, urinary magnesium, uric acid, phosphorus or oxalate. Urinary citrate was correlated with urinary calcium only in stone formers and was not correlated with urinary pH.(abridged author abst.)

ACCESSION NUMBER: 880534324 JICST-EPlus
TITLE: Clinical studies of citrate therapy on urolithiasis. Estimations of urinary citrate excretion in patients with urolithiasis and the results of treatment with sodium-potassium citrate.
AUTHOR: YASUKAWA SHU
CORPORATE SOURCE: Wakayama Medical College
SOURCE: Nippon Hinyokika Gakkai Zasshi (Japanese Journal of Urology), (1988) vol. 79, no. 4, pp. 620-628. Journal Code:
Z0766A (Fig. 5, Tbl. 3, Ref. 25)
ISSN: 0021-5287
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
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L29 ANSWER 17 OF 17 JICST-EPlus COPYRIGHT 2000 JST
TI Idiopathic **hypercalciuria** in children. Reference values for **screening**.
ACCESSION NUMBER: 870343433 JICST-EPlus
TITLE: Idiopathic **hypercalciuria** in children. Reference values for **screening**.
AUTHOR: OKADA MITSURU; YOSHIOKA KAZUO; SAKANO KAZUMI; MORIMOTO YASUO; MAKI SUNAO
CORPORATE SOURCE: Kinkidai I
SOURCE: Nippon Shonika Gakkai Zasshi (Journal of the Japan Pediatric Society), (1986) vol. 90, no. 12, pp. 2701-2706. Journal Code: F0896A (Fig. 9, Tbl. 1, Ref. 10)
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